REPORT ON THE FIFTH COMMONWEALTH MYCOLOGICAL CONFERENCE 1954

THE COMMONWEALTH MYCOLOGICAL INSTITUTE
KEW, SURREY
1954

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COMMONWEALTH MYCOLOGICAL INSTITUTE

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REPORT ON THE FIFTH COMMONWEALTH MYCOLOGICAL CONFERENCE, 1954

1. Calling of the Conference

By a resolution of the first Imperial Mycological Conference in 1924 further conferences were to be held every five years. In accordance with the resolution conferences were held in 1929 and 1934, but that for 1939 was postponed on the outbreak of war. It was not possible to hold this fourth conference until 1948. The period after the Second Great War has been very unsettled, but the Executive Council felt justified in issuing invitations in January 1953 for the Fifth Mycological Conference. The response to these invitations was most encouraging and the conference was accordingly held in London in July 1954.

2. Delegates

The following delegates attended the Conference:—

UNITED KINGDOM	Mr. F. C. Bawden Mr. W. Buddin Mr. W. C. Moore Dr. C. E. Foister Dr. J. Grainger Professor A. E. Muskett Mr. T. R. Peace Mr. G. Samuel
CANADA	Dr. W. C. Broadfoot Dr. J. W. Groves Dr. W. F. Hanna Dr. G. B. Sanford Dr. M. F. Welsh
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SOUTH AFRICA	Dr. F. C. Loest
INDIA	Dr. T. S. Sadasivan Dr. R. S. Vasudeva
PAKISTAN	Dr. Taskhir Ahmad

SOUTHERN RHODESIA	Dr. G. R. Bates
CEYLON	Mr. D. V. W. Abeygunawardene
COLONIAL TERRITORY	Sir Geoffrey Clay Mr. D. Rhind
KENYA	Dr. R. M. Nattrass
JAMAICA	Mr. H. C. James
NIGERIA	Mr. R. H. Cammack Mr. F. W. Toovey Dr. J. M. Waterston
UGANDA	Mr. D. G. Thomas
FEDERATED MALAY STATES	Mr. A. Johnston
REPUBLIC OF IRELAND	Professor R. McKay

Mr. R. M. Lister representing West African Cacao Research Institute, Gold Coast.

Mr. W. Nowell representing Imperial College of Tropical Agriculture, Trinidad.

Mr. R. D. Allen representing West African Institute for Oil Palm Research.

Dr. Lee Ling, *Observer*, Food and Agriculture Organization of the United Nations.

Visitors

Invitations to attend the Conference and take part in the discussions were issued to the staffs of Agricultural and Horticultural Research Stations, University Departments, and individual mycologists in the United Kingdom and to the representatives of various firms interested in the manufacture of fungicides and machinery. Many visitors were able to attend and they made valuable contributions to the discussions.

3. Place of Meetings

By courtesy of the Governing Body of the Imperial College of Science and Technology, meetings of the Conference were held in the Metallurgical Lecture Theatre of the Royal School of Mines, Prince Consort Road, South Kensington. In addition the Governing Body arranged for delegates and visitors attending the Conference to have meals at the College Union.

4. Government Supper to Delegates of the Commonwealth Entomological and Mycological Conferences and to Members of the Executive Council of the Commonwealth Agricultural Bureaux

Her Majesty's Government in the United Kingdom gave a supper at Hampton Court Palace on the evening of Thursday, the 15th July, to delegates of both Conferences and to members of the Executive Council of the Commonwealth Agricultural Bureaux. The Right Honourable Sir Thomas Dugdale, Bt., T.D., M.P. (Minister of Agriculture and Fisheries), presided and proposed the toast of 'The Entomological and Mycological Conferences' to which the Acting Vice-Chairman of the Executive Council, Lt.-Col. J. G. Robertson, responded on behalf of the Executive Council, Dr. E. S. Narayanan (India) on behalf of the Entomological Conference, and Dr. C. J. Magee (Australia) on behalf of the Commonwealth Mycological Conference.

5. Programme

The following programme was arranged for the Conference:

Friday, 16th July

10 *a.m.* Opening of the Conference by Lt.-Col. J. G. Robertson, Acting Vice-Chairman of the Executive Council of the Commonwealth Agricultural Bureaux.

10.30 *a.m.* THE WORK AND ORGANIZATION OF THE COMMONWEALTH MYCOLOGICAL INSTITUTE, 1948–54.

11 *a.m.* to 1 *p.m.* PLANT DISEASE LEGISLATION AGAINST SEED-BORNE DISEASES.

2.30 to 4.30 *p.m.* LOSSES CAUSED BY PLANT DISEASES IN THE COMMONWEALTH.

Saturday, 17th July

10 to 11 *a.m.* MEETING OF DELEGATES.

11 *a.m.* to 1 *p.m.* MEETINGS OF SUB-COMMITTEES.

3 to 5 *p.m.* VISIT BY CONFERENCE DELEGATES TO THE COMMONWEALTH MYCOLOGICAL INSTITUTE.

Monday, 19th July

10 *a.m.* to 1 *p.m.* CEREAL RUSTS AND THEIR CONTROL.

2.30 to 4.30 *p.m.* ERADICATION OF ESTABLISHED PLANT DISEASES.

Tuesday, 20th July

10 *a.m.* to 1 *p.m.* FUNGICIDES (WITH SPECIAL REFERENCE TO ANTIBIOTICS, SYSTEMIC FUNGICIDES, AND CONTROL OF BLACK POD OF CACAO).

2.30 to 4.30 *p.m.* (*a*) TOBACCO DISEASE PROBLEMS IN SOUTHERN RHODESIA.
(*b*) OIL PALM DISEASES.
(*c*) BLISTER BLIGHT OF TEA.

Wednesday, 21st July

Visit to East Malling Research Station.

Thursday, 22nd July

10 *a.m.* to 1 *p.m.* VIRUS DISEASES (WITH SPECIAL REFERENCE TO VIRUS DISEASES OF FRUIT TREES AND STREAK MOSAIC OF WHEAT).

2.30 to 4.30 *p.m.* VIRUS DISEASES (CONTINUED, WITH SPECIAL REFERENCE TO CITRUS TRISTEZA AND LIME DIE-BACK, AND STUNTING DISEASE OF SUGAR-CANE).

Friday, 23rd July

10 *a.m.* to 1 *p.m.* MEETINGS OF SUB-COMMITTEES.

2.30 to 4.30 *p.m.* FINAL PLENARY SESSION.

EXCURSIONS

During the week after the Conference visits were paid to the following research centres:

Monday, 26th July: Agricultural and Horticultural Research Station, Long Ashton.

Tuesday, 27th July: National Institute of Agricultural Engineering, Silsoe, and Forest Products Research Laboratory, Princes Risborough.

Wednesday, 28th July: Rothamsted Experimental Station.

Personal invitations were sent to all delegates by Plant Protection Ltd. to attend a field demonstration on Thursday, 29th July. Many of the delegates took advantage of the opportunity to see demonstrations of crop spraying machinery at Fernhurst.

6. Opening of the Conference

The Conference was opened by Lt.-Col. J. G. Robertson, B.S.A., F.R.S.A., Acting Vice-Chairman of the Executive Council of the Commonwealth Agricultural Bureaux, at 10 a.m. on the 16th July. Colonel Robertson said that he was very glad to be the first to welcome the delegates and visitors to this the Fifth Commonwealth Mycological Conference.

He had had the privilege of officially opening the Fourth Commonwealth Mycological Conference in 1948, as Chairman of the Commonwealth Agricultural Bureaux. On the present occasion he was acting for the Chairman, Mr. W. F. C. Morton, of the Union of South Africa, who was unfortunately unable to be present but extended to all delegates his best wishes for a very successful conference.

Lt.-Col. Robertson explained the constitution and functions of the Executive Council of the Commonwealth Agricultural Bureaux, pointing out that it is financed by contributions from the various Commonwealth Governments in proportion to their interests and requirements, each Dominion or body having equal status, although each does not contribute the same amount financially.

He paid tribute to the foundation work of Sir David Chadwick, Secretary of the organization from its inception until 1946 (who died earlier this year), to Sir Herbert Howard and his assistants, who have ably continued the work since then, and to the present Director and Assistant Director of the Commonwealth Mycological Institute.

He said that in addition to the three institutes of Entomology, Mycology, and Biological Control governments were considering the establishment of two or three more. He would not list the names of the Directors or Consultant Directors, suffice to say that they and their assistants were all skilled in their work and efficient in the performance of their duties.

The Executive Council controlled the expenditure of a large amount of money, which was increased by contributing governments after the 1950 Review Conference to over £228,000 a year. Still more could be used with advantage, but the Executive Council had to be reasonable in their requests.

The present Conference was mainly concerned with the work of the Commonwealth Mycological Institute, and he felt sure that it would be successful in its deliberations.

The history of the Commonwealth Mycological Institute was a fairly long one as it was founded in 1921 and was senior to all the bureaux. Its first Director was Sir Edwin Butler (1921–35); the second Mr. S. F. Ashby (1935–40), who died this past spring; the third and present Director, Dr. S. P. Wiltshire, since 1940.

After repeating his cordial welcome to the delegates Lt.-Col. Robertson declared the Conference open.

7. The Work and Organization of the Commonwealth Mycological Institute

Following the opening address by Lt.-Col. Robertson, the Director reviewed the work of the Commonwealth Mycological Institute since the last Conference; his review is published as APPENDIX I to this report.

8. Appointment of Committees

Two sub-committees were then appointed:

(*a*) to consider the work and needs of the Institute, composed as follows:

Mr. W. C. Moore
Dr. W. F. Hanna

Dr. C. J. Magee
Dr. E. E. Chamberlain
Dr. F. C. Loest
Dr. R. McKay
Dr. R. S. Vasudeva
Mr. D. V. W. Abeygunawardene
Dr. Taskhir Ahmad
Dr. J. M. Waterston
Dr. R. M. Nattrass
Dr. G. R. Bates

(*b*) to draft resolutions, composed as follows:

Dr. W. C. Broadfoot
Mr. G. Samuel
Mr. F. W. Toovey

9. General Discussions of the Conference

A summary of the papers and discussions of the Conference is given as APPENDIX II to this Report.

10. Visits to Research Stations

On Wednesday, 21st July, most of the delegates visited East Malling Research Station, Kent, by kind invitation of the Director, Dr. F. R. Tubbs, who welcomed them and gave an address on the history of the station and work in progress. Dr. R. V. Harris, head of the Plant Pathology Department, then outlined the research programme in plant pathology, which was followed by a talk on *Verticillium* disease of hops.

After lunch delegates viewed some of the work being performed in the laboratories and field plots, and were able to discuss problems with members of the research staff.

Arrangements were also made for parties to visit other research stations and institutes during the week following the Conference, as indicated in the programme.

11. Report of the Technical Committee on the Work and Needs of the Commonwealth Mycological Institute

The following report was unanimously adopted at the Plenary Session on Friday, 23rd July.

1. We were appointed on 16th July at the opening session of the Conference to consider the work and needs of the Institute. Dr. Wiltshire attended the three meetings we held and greatly assisted us when discussing the progress made in implementing the recommendations of the 1948 Conference, and the possible expansion of present lines of work.

2. We desire first to record our warm appreciation of the continued services rendered by the Institute, under the able guidance of Dr. Wiltshire, to mycologists and plant pathologists throughout the Commonwealth. We also express the hope that sufficient financial provision can be made to ensure that these services are fully maintained to meet the increasing demands made on the Institute by Commonwealth countries.

3. *Handbooks.* In considering the progress made since the last Conference we note that a manual on rice diseases has been published and that others on sorghum diseases and on tobacco diseases in Africa are in hand. We understand that it has not been possible to obtain completion of the promised handbook on diseases of cacao or to make arrangements for preparing much needed books on the diseases of sugar-cane, cotton, coffee, coconut and other palms, tropical cereals, and miscellaneous food crops. We strongly urge that further efforts be made to encourage the preparation of handbooks on these subjects, and to assist in achieving this object we recommend that consideration be given to the payment of honoraria or royalties to authors.

4. *Monographs.* It is gratifying to report rather more progress with the series of monographs on groups of fungi and on particular diseases. Monographs have been published on Leaf Curl of Cotton, Indian Smuts, British Smut Fungi, and on the genus *Periconia*.

5. *Microfilms.* We understand that the microfilm service, although not in great demand, is still available, but that reproduction of standard publications by micro-cards is more convenient and practical for general use. The purchase of micro-card readers is desirable but not essential, because a binocular microscope can serve the same purpose.

6. *Culture Collection.* At the Commonwealth Conference on Culture Collections it was recommended that the Institute should maintain a central collection of fungus cultures. We were gratified to note the steady progress made in building up this collection and to learn that an up-to-date catalogue and supplements are available. We note with appreciation that this work has been greatly facilitated as a result of a substantial grant from the D.S.I.R. We feel, however, that it is desirable to bear in mind that further financial provision may be necessary as the collection grows and becomes more widely known and utilized.

7. *Loose-leaf 'Working Sheets'.* We note with regret that owing to shortage of staff and funds it has not been possible to begin the issue of loose-leaf sheets giving summary descriptions of pathogenic fungi. Sample sheets circulated among plant pathologists have met with the warmest commendation, and we most strongly urge that the necessary additional funds and staff be provided without delay

to develop this project, for we are convinced that the sheets would soon be in great demand. We understand that a start has been made with 25 sheets dealing with species of *Phytophthora*.

8. *Exsiccata.* We observe with satisfaction that a number of exsiccata have been prepared and packeted ready for exchange with suitable institutions at the discretion of the Director. We recommend that equipment for the reproduction of labels be provided, as labelling by hand is excessively laborious.

9. *New Building.* We inspected the plans and viewed the work in progress on the new building, and are pleased to learn that it is expected to be completed by June 1955.

10. *Broadsheet.* We were given details and shown an outline of the broadsheet of which it is hoped to produce the first number in January 1955. It will be a periodical publication and will include technical information in some instances from territories where national publications do not exist, as well as personal and other interesting items supplied by correspondents in the various territories of the Commonwealth.

11. *Training in Taxonomy.* The vital importance of taxonomic work on fungi in the solution of Commonwealth plant disease problems was re-emphasized during our discussions and at the same time the need was stressed for additional posts in taxonomy at the Institute as well as for facilities to enable mycologists to be given advanced training in taxonomic methods. The Director assured us that he would be only too pleased to help in providing such training for keen and promising mycologists holding appointments in the Commonwealth, and said that facilities in the new building would be adequate for this purpose.

12. *Staff Needs.* Having reviewed the increasing work of the Institute and made certain recommendations that will still further increase its commitments, we turned our attention to staff needs. We are concerned at the prospect that the approaching retirement of several members of the staff who are recognized authorities in certain specialized fields of mycology will leave gaps which will be very difficult to fill. It is recognized that a competent taxonomist requires many years of training, and that suitable candidates are rare. It is therefore highly important to ensure as far as possible a succession of suitably trained officers. It is partly with this object in view and partly to meet further commitments that we recommend the establishment of four extra posts in Grade A/B. This should be sufficient to cope with increasing taxonomic work, to provide for special taxonomic studies in important groups of fungi, and to promote progress in the production of the 'Working Sheets' referred to above. Such expansion as we envisage will, of course, involve the appointment of further ancillary staff.

We regard it as of the utmost importance fully to maintain the high standards set from the beginning by the *Review of Applied Mycology*, and consider that the present number of abstractors in the Institute is too small to cope with the enormous increase in plant pathological literature in recent years, a trend which is likely to continue. We therefore recommend that financial provision be made for two additional abstractors.

12. Resolutions

The following resolutions were adopted by the Conference:

1. The Conference is of the opinion that since the transfer of planting material from one part of the Commonwealth to another involves a risk of transferring serious plant diseases, it is essential that the service provided by the present plant quarantine greenhouse at Kew should be maintained, but it considers that the method of operating the service needs reviewing.

2. Since the provision of disease-free seed is an important means of safeguarding the health of crops, the Conference considers there is need: (i) for developing standard methods for the rapid and accurate determination of as many seed-borne diseases as possible, and (ii) for surveys to be made to discover areas where seed-borne diseases of the various economic crops are at a minimum, with the object of encouraging the production of commercial seed of the highest standard of health.

3. The Conference is of the opinion that there is need to develop uniform methods of recording the losses due to plant diseases in the Commonwealth and that there is also a need for experiments to determine the reduction in yield and quality of crop plants associated with various degrees of infection.

4. In view of the successful eradication of several major diseases from Australia by the vigorous application of sound plant pathological methods, and the measure of success obtained by using similar methods in New Zealand and elsewhere, the Conference urges that all countries should give careful consideration to the possibility of applying such methods, especially to diseases newly introduced or still limited in their distribution.

5. The Conference recommends that, with a view to limiting the spread of virus diseases of fruit trees and ensuring that new orchard plantings are established with as healthy material as possible, there is need for increased work on the indexing of nursery stocks of all important tree fruit varieties subject to virus diseases.

6. The Conference considers that planting material of tree and soft fruits transferred from one country to another should, as soon as practicable, be limited to certified material derived from stocks indexed for freedom from virus diseases.

7. The Conference recognizes the advantages to plant pathologists of being able to obtain rapid and precise identification of bacterial plant pathogens, particularly in cases where a new bacterial disease is suspected in an area and where early identification of the pathogen is essential to justify efforts to limit or eradicate the disease. The Conference therefore recommends that the Permanent Committee of the British Commonwealth Culture Collections should be asked to consider the problem and to review the facilities required for rapid identification of bacterial plant pathogens, including the possibility of establishing at some central laboratory a collection of specific absorbed sera and/or of bacteriophages.

8. The Conference is of the opinion that there is urgent need for more taxonomic work in mycology to provide a sound basis for the identification of pathogens of importance in plant pathology. They concluded that the only way in which such work can be encouraged is by the creation of further posts in taxonomic mycology. They stressed the need for the creation of such posts, both at the Commonwealth Mycological Institute and at other suitable institutions and recommended that the Executive Council of the Commonwealth Agricultural Bureaux should draw this matter to the attention of appropriate authorities.

13. Votes of Thanks

A vote of thanks to the authorities of the Imperial College of Science and Technology and to the Imperial College Union for the use of their rooms for the meetings and for the facility granted to delegates and visitors of taking meals in the College refectory was passed. Votes of thanks were also passed to the Director and staff of the Commonwealth Mycological Institute for their work in connexion with the Conference, to the Resolutions Committee, to the Chairman, Lt.-Col. Robertson, to East Malling Research Station for the excellent arrangements made for the benefit of the delegates; also to Long Ashton Agricultural and Horticultural Research Station, the National Institute of Agricultural Engineering, Rothamsted Experimental Station, and to the Forest Products Research Laboratory, Princes Risborough, for kindly arranging visits for the delegates during the following week.

APPENDIX I

The Director's Review of the work of the Institute 1948-1954

The six years since the Conference last met have been years of difficulty for the Institute. For the first three years we were working on a basis of about £13,000 from Commonwealth Governments and this meant the most stringent financial retrenchment, which seriously impeded our work. The Review Conference of 1950 recommended increased contributions from governments and as a result they were raised to a total of £27,000 in 1952-3, and this eased the financial strain from April of that year.

In February, 1950, an opportunity occurred of acquiring the adjacent properties 59, 61, and 63 Kew Green and these were purchased to provide a site for the new building so strongly recommended by the last Commonwealth Mycological Conference, 1948. Plans were prepared at short notice and circulated to members of the Review Conference in 1950 who endorsed the proposed new building. The financing of the new building raised problems of considerable magnitude but it is very heartening to know that all obstacles were overcome and the contract for the new building was placed in April last. The building is now in course of erection and is expected to take a little over a year to complete. In the meantime four rooms in 61 and 63 Kew Green became available and have provided valuable accommodation for the abstracting and publications staff.

A feature of the past six years has been the rapid progress made in plant pathology and mycology throughout the world. The accumulation of information on our subjects has been amazing and the mass of literature published has increased accordingly. There is no doubt the world is becoming more and more conscious of the serious losses that are caused by plant disease and many countries that until recently had no plant pathologist have now appointed one or more. To cope with this volume of information now being amassed at such speed it is more important than ever to have it properly abstracted and documented and adequate abstracting services provided. It is against this background of remarkable development that the work of the Institute must be reviewed.

Our main abstracting journal, the Review of Applied Mycology, has increased the number of abstracts published from about 1,800 a year to about 2,300. Last year we printed 704 pages apart from the index as against 592 pages in 1952, and further increases are anticipated. Dr. J.C. Hopkins has been recently appointed to assist in the work and further staff will no doubt be needed. The Review is a most important part of our activities and it is essential to maintain the standard of our abstracting and keep up-to-date with the work. The cost of printing has increased nearly 50 per cent. since 1948, but this has been mitigated by an increase of 10s. in the subscription price.

The increase in incoming literature has also affected our other publications.

The Index of Fungi has been published regularly at approximately six-monthly intervals, but the numbers of new genera, species, varieties, and new names have increased considerably. In 1940-3 the first 7 parts of Vol. I of the Index comprised 86 pages, in 1950-3 the corresponding figure for the first 7 parts of Vol. II was 141 pages. Subscribers have increased from 176

in December, 1948, to 260 in 1953, a good number for such a specialized journal. A cumulative index for the ten years 1940-49 was compiled but could not be printed until this year owing to lack of funds. It is some satisfaction to us that it has at last appeared. A cumulative index is being compiled for Vol. II of the Index of Fungi in readiness for the completion of that volume.

During the year 1950-51 Petrak's list of new species for 1936-39 was completed by Dr. Petrak at our suggestion and published by the Institute. In addition his list for 1932-35 was made available and since then his other six lists covering the years 1920-31 have all been republished, so that we have now a more or less complete record of new fungus names from 1920 to date. For the years before 1920 Saccardo's Sylloge fungorum serves for the time being. A cumulative index for Petrak's lists is well advanced and I hope it will be published within the next two years.

For many years a project for the issue of microfilms containing diagnoses of new species from the original journals has been our objective. We had made considerable progress when the disastrous flooding of our book store which I shall have to refer to later put a stop to the work until recently. Instead of microfilms we finally decided to issue microcards of the diagnoses as being much handier to use. Upwards of 90 journals have generously granted us permission to reproduce diagnoses published in their pages. The preparation of the first set of 25 cards is now well advanced and it is hoped the cards will be available in the autumn. An index of the species concerned is given with each set of cards. The cards can be read by means of a binocular microscope but microcard readers are available from the United States.

The "Bibliography of systematic mycology" has appeared at intervals covering the years 1943-1952.

The "Distribution maps of plant diseases" have regularly appeared at the rate of two a month and to date some 290 maps have been issued. A cumulative index of Maps 1-240 for the ten years 1942-51 was issued free to subscribers in 1952. The number of subscribers have been maintained round about 250 indicating that the demand for these maps is now satisfied. In connexion with the world reporting service of F.A.O. it was agreed by the Colonial Office that records of plant diseases from Colonial Territories should be channelled through the Institute.

'Mycological Papers' have appeared at irregular intervals during the period under review and to date 57 papers have been issued. These Papers record the results of research carried out at the Institute or in the Commonwealth and meet a need not provided for elsewhere.

The Annotated Bibliography of Medical Mycology has been continued and is gradually becoming better known. A grant-in-aid of £250 from the Medical Research Council towards the cost of the publication was received during the years 1951-53. Subscribers total 255. The field surveyed has always included veterinary mycology and with the increasing development of that science it was decided to rename the publication the Review of Medical and Veterinary Mycology. The collaboration between medical men and mycologists has been fruitful and it is true to say the subjects of medical and veterinary mycology have been greatly stimulated in this country as a consequence.

Since the last Conference the Institute has issued a number of books in accordance with the Resolution 7 of the last Conference. 'The Manual of Rice Diseases' by Dr. Watts Padwick was issued in 1950 and has been well received. It was the first book on the subject and has proved of great value to tropical plant pathologists throughout the world, bringing together as it does the knowledge of the subject available at the time of publication.

A monograph on "British Smut Fungi" by Dr. Ainsworth and Miss Sampson was published in 1950. The work comprises a general account of the group and a new systematic treatment of all the smuts recorded in Britain. The last English book on the smuts was Plowright's monograph, published in 1889. The book is a valuable contribution to our literature on this important group of plant pathogens.

In 1951 a monograph of the''Leaf Curl Disease of Cotton' by Dr. S.A.J. Tarr was published by the Institute. This monograph has a limited appeal to plant pathologists, as there are comparatively few interested in the cotton crop and fewer still in cotton leaf curl. Sales have therefore been somewhat restricted but the book has been favourably received and gives an authoritative account of one of the major diseases of cotton.

A third monograph dealing with the Ustilaginales of India by the late Dr. Mundkur and Dr. Thirumalachar was issued in February 1952. It brings together information on all the smuts found in India and should prove of great value to plant pathologists and mycologists not only in that country but elsewhere in facilitating the recognition of species of these fungi, many of which are important pathogens.

The first edition of 1,500 copies of Dr. Bisby's 'Introduction to the Taxonomy and Nomenclature of Fungi' was exhausted in February 1952. A second edition had to wait for the appearance of the revised International Rules of Botanical Nomenclature but was issued in January 1954. It included an extra part, 'Notes on classification'. This book has proved acceptable to students and others and sales have been good.

An event of serious importance to our publications section was the flooding of our publications store in 63 Kew Green on the night of 31st January to 1st February, 1953, the night of the great flood and tempest that did such damage to the East Coast and Holland. The tidal wave swept over the bank of the Thames and up the garden and flooded the cellar in a few minutes. With the willing help of the junior staff and some casual labour some 12 tons of books were removed from the cellar two days later and sent to the Oxford University Press who very kindly undertook to dry them. The disaster threw a heavy burden of additional work on the Institute at a time when we were ill prepared to undertake it, but in spite of it all it is very satisfactory to note that sales of publications and cultures reached a record total of £7,646. Much still requires to be done, but reprinting has commenced and it is hoped ultimately to maintain all our publications in print. Happily we had reserve sheets at the Press for all our books except the Dictionary of the Fungi and Dr. Bisby's Introduction, but the type of both of these is kept standing. All our books are now available again. We shall probably feel the effects of this flooding for several years and there is no doubt it has put us back in our work very seriously.

One result of the flooding was that about 1,500 copies of the third edition of the Dictionary were lost, and instead of merely reprinting it the opportunity

was seized of bringing out a fourth edition of this useful work.

The Culture Collection has made rapid progress since the last Conference. The number of cultures maintained in the Collection increased from just over 500 to 1,783, the number of cultures distributed per annum from 203 to 1,223 and receipts from £119 to £279. In this work Major Dade has the help of two technical assistants and recently Dr. Agnes Brown has been appointed to deal with species of Penicillium, the identification of which she now undertakes. The collection has been hampered severely by lack of accommodation but this is being rectified in the new building. Very considerable demands are made on the collection by industry and it is a pleasure to acknowledge the assistance of the Department of Scientific and Industrial Research in making a grant recently raised to £2,000 in aid of this work.

During 1952 an important development was the establishment by the Colonial Office of a Pool of Plant Pathologists attached to the Institute under a scheme financed by the Colonial Development and Welfare Fund. The aim of the Pool is to provide plant pathologists to undertake the investigation of specific problems in Colonial territories where trained staff is not available for this work. The first appointment was Mr. R. A. Altson, who had retired from the position of Pathologist in charge of the Pathological Division of the Rubber Research Institute of Malaya. The Government of Cyprus asked for his services in regard to a disease of broad beans which was estimated to be causing a loss of £10,000 per annum. After studying the relevant local reports and literature Mr. Altson left for Cyprus in January, 1953. The investigation has not been an easy one and there are various points that might be followed up in future but after a long series of inoculations Mr. Altson has succeeded in establishing that Fusarium oxysporum and its var. redolens, repeatedly isolated from diseased plants, are the cause of the disease. The broad beans grown in Cyprus are a Seville variety with a very short pod and Mr. Altson obtained through a firm of wholesale seed merchants in London seed of long-podded Seville varieties from French Morocco, two of which have been found to be less susceptible to the disease than the locally grown variety. Further testing of varieties for resistance and breeding is evidently required but there is hope that a satisfactory control may be discovered.

In Cyprus the broad beans are sometimes eaten raw and a disease known as favism is prevalent among the population and is attributed to this cause. Mr. Altson has found a species of Penicillium growing on the unopened cotyledons of the healthy beans, an unusual site, and the phenomenon may be worth further investigation with reference to favism.

The new building now being erected provides ample accommodation for the library, the herbarium, the culture collection, visitors' rooms, dark room, and storage for publications. The plans were worked out in great detail and the building should serve the needs of the Institute admirably. It is a pleasure to me to acknowledge the excellent work of the architects, Messrs. Waterhouse and Ripley, and their valuable assistance in overcoming the many difficulties that have been encountered.

Turning now to the identification service, the other major activity of the Institute, I have to report ever increasing numbers of specimens received for determination. At the last Conference the specimens in the herbarium indexed and not indexed numbered about 36,000 (including 6,000 not indexed) now we are approaching 60,000, an increase of over 60 per

cent. The indexing of the herbarium is now complete except for the rusts, and at present we are dealing with about 4,000 incoming specimens a year. Without the reorganization of the herbarium reported to the last Conference it would have been impossible to deal with this very considerable number. The herbarium technique we have developed is proving more and more valuable as the years go by and is being developed still further. All accessions to the culture collection are represented in our herbarium by dried cultures of each as received, so that if saltation takes place in culture we have material of the original culture for reference. Our techniques for drying cultures, for sealing slides, and for various culture operations have been improved during the last six years. We hope to demonstrate some of them to Delegates to-morrow at the Institute.

The Institute suffered a great loss in the departure of Mr. S.J. Hughes to Ottawa, especially as he had been trained intensively in our methods with a view to his following Mr. Mason in due course. Happily we had Dr. Ellis to take Mr. Hughes's place, but it was difficult to find anyone with the requisite taxonomic ability to recruit for the identification service. After some delay we succeeded in securing Mr. Booth for the work. Of course, Dr. Brown, who was appointed to study Penicillium and help in the culture collection, is being trained in our herbarium methods, as this is indispensable for her work.

Besides the routine work of the Institute, we are asked to advise the Colonial Office regarding disease problems from time to time. Of those encountered during the last six years I may briefly mention the following:

Maize rust (Puccinia polysora), previously known only from the American continent, appeared in 1950 in West Africa, where it caused great loss of crop. This important determination was of basic value in formulating research on the disease and a maize rust research unit was established in Nigeria. Mr. Cammack will, I hope, give an account of this work during the Conference, so I need only say that the rust has spread throughout central Africa and extended at least as far south as Zululand and east to Madagascar, Mauritius, and Réunion and now threatens India, Ceylon, and possibly Australia and south-east Asia.

The mystery of sudden death of cloves in Zanzibar that has baffled all sorts of people for the last 30 years has been solved at last. Dr. Nutman and Miss Roberts have established the cause to be a new species of Valsa which they named V. eugeniae; they also found that sudden death is confused with another disease they named die-back caused by a new species of Cryptosporella which they described as Cryptosporella eugeniae. The determination of the etiology of these two diseases led to effective control measures being devised.

A serious root disease of pepper (Piper nigrum) was reported from Sarawak in 1953. From a preliminary survey by Mr. N. Robertson of the Botany School, Cambridge, it appears that the disease is similar to and probably the same as that attacking pepper in Indo-China. The cause of the disease in Indo-China is attributed to the joint action of nematodes and fungi but Mr. Robertson does not agree altogether with this view. The disease is a very virulent one and until pathogenicity is decisively proved one suspects that the real pathogen has not yet been discovered.

Blister blight of tea (Exobasidium vexans) is still prevalent in Ceylon and has extended its area to Sumatra, Java, and Malaya. In Ceylon the

disease is considerably controlled by modified cultural methods and by spraying. In the wetter regions of Indonesia and Malaya control is more difficult but not impossible.

In Malaya the serious threat to the rubber-growing industry of the possibility of the introduction of the deadly Dothidella ulei has been appreciated by the Rubber Research Institute and plans are in hand to mitigate such a catastrophe by all possible means. This is a very wise development. If major outbreaks of disease are to be controlled, preparations beforehand are indispensable to be effective. Throughout the world several major threats to national crops easily come to mind and one hopes that the general disinclination to prepare for the worst will not lead to disasters such as we have had in the past.

Another development of modern plant pathology is the large-scale eradication of disease. At the last Conference Dr. Hopkins gave an account of his successful efforts to keep Southern Rhodesia free from tomato spotted wilt. The classic example of the eradication of citrus canker from the Northern Territory of Australia and from the Union of South Africa showed the way for the Gulf States of the United States to do the same. By the strict and persistent application of control measures against sugar-cane diseases in Queensland, downy mildew has been practically eliminated, Fiji disease reduced to an incidence of a few hundred stools, and gummosis reduced to secondary importance. We are happy to have Dr. Magee with us to-day and we hope to hear more about this work in our discussions and everyone will hope that by modern techniques it will be feasible to eliminate some of the devastating diseases that take so great a toll of some of our crops.

In the realm of fungicides important advances have been made in the discovery and formulation of new complex organic fungicides, in the use of low-volume sprays, the development of more efficient spraying and dusting apparatus, and in the study of systemic fungicides, which we now know do exist, and the search for those applicable to plant disease control is now actively being pursued with very promising results. Furthermore, the use of fungicides in the tropics is now spreading rapidly in the changed economic conditions of to-day. Six years ago few plant pathologists believed that spraying or dusting against diseases of tropical crops was feasible. Now plant protectives are regularly used against a number of diseases, the most recent successes being the seed disinfection of cotton lately adopted in Uganda with great profit and its probable use, as a result of Mr. Thorold's studies, against black pod of cacao in West Africa. A marked change has therefore come over the whole phytopathological scene and there is more hope of plant disease control in the tropics than ever before. Let us hope it will be utilized to the full.

From this very brief survey it is safe to conclude that the progress made in the plant pathology in the last six years is likely not only to continue but that the tempo of advance is likely to increase rather than diminish. It is evident that the Institute must keep abreast of all these developments and adequate funds will be required for this. It is not to be expected that money for the work will be forthcoming unless legislators, administrators, and all others concerned in the growing of agricultural crops are apprised of the magnitude of the problems to be tackled and of the importance of the work. To supply information on all aspects of plant disease is the duty of plant pathologists, and the discussions to be held at this Conference will, perhaps provide material of use to this end. Moreover, it is desirable to take a broad view of the basic requirements of plant pathologists and not restrict

the work to that immediately and directly connected with plant diseases. Unhappily, such problems are apt to be regarded as outside our field of investigation and in the past I have often wished the Institute had some endowment to finance urgent work and special projects not provided for in any other way. A careful consideration of our present needs in relation to the work we do, will, I believe, lead to the conclusion that our work is productive and well worth while paying for.

In conclusion, I would like again to acknowledge our indebtedness to the Director and Staff of the Royal Botanic Gardens, Kew, including Dr. Dennis, Cryptogamic Botanist, and Dr. Metcalfe, Assistant Keeper of the Jodrell Laboratory, for the help so kindly given through the past six years.

I would also like to pay a tribute to my own staff for their loyalty and patience under very crowded working conditions for some years under severe financial stringency and under great pressure of work. I hope better times are in store.

Finally I would refer to the death of Mr. S. F. Ashby, the second Director of the Institute, who died on 6th March last, after a distinguished career as plant pathologist and mycologist. An obituary notice appeared in Nature and a few copies are available to Delegates who would like one.

APPENDIX II

Commonwealth Mycological Conference, July, 1954

MINUTES OF THE SCIENTIFIC DISCUSSIONS OF THE CONFERENCE

Meeting: Friday, 16th July, 11 a. m.

Chair: SIR GEOFFREY CLAY

Subject: Legislation against seed-borne diseases

Paper: Plant disease legislation against seed-borne diseases

by W. C. MOORE
(Ministry of Agriculture & Fisheries,
Plant Pathology Laboratory, Harpenden)

Most countries now impose health requirements for the importation of planting material such as nursery stock, bulbs, and potatoes, but this is not true for seeds. Scrutiny of the plant import regulations of 90 countries or territories made by one of my colleagues, Mr. J. J. Baker, revealed that about one-half of them do not ask for any form of health certificate with seeds, though many require an import permit to be obtained for some or all seeds.

Of the other half, 28 demand a general health certificate for all seeds, and sometimes an import permit, and 14 require a similar certificate for some seeds.

The requirements as a whole comprise all sorts of combinations of general and special health certificates, import permits, and other conditions. Fortunately, I do not need to enumerate them or to try and justify or impeach them. For my purpose it is sufficient to state that about 20 countries prohibit altogether the importation of certain kinds of seeds, such as tobacco, cotton, and clover; that various countries reserve the right to inspect some or all seed consignments on arrival; that a few countries require certain seeds to be declared free from dodder, broom-rape, or other weed seeds; and that there is an increasing tendency for countries to impose requirements designed to prevent the introduction of particular diseases. For instance, at least five countries now call for declarations about the freedom of tomato seed from Corynebacterium michiganense, the cause of bacterial canker in that crop, and other diseases made the subject of special requirements by one country or another include bacterial wilt (Xanthomonas stewarti) and smut (Sorosporium reilianum) of maize, pasmo (Sphaerella linorum) of flax, bacterial blight (Pseudomonas pisi) of peas, and black rot (Xanthomonas campestris) of cabbage.

Those countries which ask for a general health certificate accept from England and Wales one which states that "a representative sample of the consignment..... has been thoroughly examined at the Ministry's Plant Pathology Laboratory, Harpenden..... and has been found or is believed to be free from injurious plant diseases and dangerous insect pests." I have often stressed the imperfections of such a certificate, but it is the best we or anyone else can provide if the demand is made. It is quite trustworthy for certain well-known seed-borne diseases where a simple test is available. But with many ornamental and vegetable seeds we do not know how to test for

parasites that might be carried on them, and because of the time factor can do no more than take a rapid look at a small sample under a low-powered microscope. Under these conditions the certificate is often worth little more than the paper it is written on.

So far I have been dealing solely with phytosanitary regulations imposed by governments. There is, however, another form of health certification that we need to bear in mind. I refer to the orange and blue forms of the International Seed Analysis Certificates used in transactions between buyers and sellers. These certificates, which are issued under the auspices of the International Seed Testing Association (I. S. T. A.), relate primarily to the purity and germination of seed samples sent for testing to Seed Testing Stations. But there is provision on them for a report on the sanitary condition of the seed. At the last meeting of the Association held in Dublin in May 1953 a new set of International Rules for Seed Testing* was adopted, and these Rules must be followed when orange or blue international certificates are to be used. The Rules include a short paragraph emphasizing the importance of the health condition of the seed. But the Association wisely refrained from attempting to lay down hard and fast rules or recommendations about methods to be followed in testing for health and made it clear that as far as health was concerned the certificates would relate only to the results of examination for specific organisms as and when such a report was requested by the sender. In an Appendix to the new Rules there is a memorandum giving general guidance to seed analysts about routine methods for detecting seed-borne organisms, together with a very useful basic list of these organisms.

To complete the picture of the present position I ought to refer briefly to the Report of the Horne Committee on Qualitative Control of Seeds, appointed in October, 1949 to consider ways and means of introducing practical methods of qualitative control of seeds used in the United Kingdom, whether home produced or imported. The Committee paid some attention to the question of seed-borne diseases, recognized that a better knowledge of seed-borne organisms and their detection is an essential pre-requisite for effective legislation, and drew attention to the urgent need for further research into methods of detecting seed-borne organisms. They also listed nearly fifty seed-borne organisms capable of adversely affecting food production.

Against this background of insufficient knowledge, and with the object of providing adequate safeguards against the introduction with seed of important diseases and pests into new territories, without involving Plant Protection Services in time-consuming, wasteful work, I would like to put forward a few suggestions for possible future action. They will at least serve as a basis for discussion.

In the first place I would plead that countries should avoid demanding health certificates for each and every kind of seed. In the present state of our knowledge they serve no real purpose and waste a good deal of time. Health certificates covering nursery stocks and bulbs can and should provide some assurance to the importer that he is receiving goods of reasonably good quality. With seeds the buyer can rely for general quality on the international orange certificate covering purity and germination, and if he wants specific assurances about health he can get them from the same source. There may be a case for requiring a general health certificate with large

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*see Proc. Intern. Seed Testing Assn., 18, 1953, 1-69.

bulk consignments of seeds, but certainly not for packeted seeds destined primarily for garden use.

Instead, each country would be well advised to prepare a list - and as short a one as possible - of those injurious seed-borne organisms which are unknown or rare in its territory. Having done so, the next step would be to lay down reasonable health requirements for the importation of each kind of seed that might carry the organisms. Such requirements ought always to have a scientific basis and they will not necessarily involve seed testing in the laboratories. Sometimes it may be necessary, or desirable, or more convenient, to require a declaration that the crop from which the seed was gathered has been inspected and found free from a given disease; necessary because no other method is possible, or desirable until a suitable laboratory method can be devised. Seed-borne virus diseases, loose smuts of cereals, and bacterial blight (Pseudomonas pisi) of peas may come into this category. At times it may be sufficient to ask for a declaration that such and such a disease is unknown in a particular country or area, but where an 'area' requirement is imposed, it will greatly help the exporting country if the area is defined in terms of field, nursery, farm, parish, county, or province, etc. For certificates based solely on laboratory testing the aim should be to employ methods that have been accepted internationally, so that each country will know precisely what is meant by the statement on the certificate. It helps the exporting country if alternative declarations are provided, but requirements must necessarily be concise. Consequently, as conditions vary so much from country to country and it is impossible to cover everything in one or two short phrases on a certificate, I think that by far the most satisfactory arrangement is for the technical officials in the importing and exporting countries to come to a mutual understanding about the precise interpretation of a requirement.

It is, of course, one thing to recommend the use of internationally accepted methods, and quite another to say what these methods should be. This is where I feel the Seed-Borne Diseases Committee of the International Seed Testing Association could play an important part. This or some other body, having been provided with a master list of economically important seed-borne diseases about which the various countries are specially concerned, could without much difficulty distinguish between those for which one or more laboratory methods are already available, and those for which no method has yet been devised. For instance, suitable methods have already been worked out for seed-borne diseases of oats and flax, as well as a number of diseases of other crops. For each such disease a sheet could be prepared giving the name of the causal organism, together with full details of the recommended method or methods of detecting it in seed samples. International agreement to the adoption of the methods for use in providing phytosanitary certificates could then be sought. The organizing body might also encourage and sponsor research on those diseases for which there is as yet no reliable method of testing the seed, with a view to devising methods internationally acceptable. In all such work it is important to ensure that the test is relatively simple and rapid, and one that takes no longer to do than testing for purity and germination. Before finally selecting a method it would be advisable, by a system of referee samples, to make sure that comparable results are obtained by workers at different stations when testing the same sample of diseased seed. I realize that all this means a long term project, but it is high time something was done to tackle the problem. Meantime, a good deal could be accomplished by closer liaison between the technical officials in importing and exporting countries.

Having agreed on methods, who would be responsible for applying them in satisfying governmental requirements? In my view health certificates for seeds should be issued only by an official Plant Protection Service or Department of Agriculture, but I see no objection in principle to the issue being made on the basis of a seed test carried out at an authorized Seed Testing Station. Indeed, it would be an advantage if the phytosanitary certificate and the international orange certificate (so far as it concerns health) were based on the same methods of testing, and were worded similarly, even though tolerances might be permissible with one and not with the other. This view, however, implies also recognition of a principle that testing for health is specialized work to be carried out, not by the traditional analysts who deal with purity and germination, but by a special staff trained and supervised by plant pathologists. Once the method has been devised routine examination can be carried out effectively by trained members of the Experimental Officer and Senior Assistant (Scientific) grades.

A fortnight ago I had an opportunity of putting forward these suggestions to a meeting of a small Working Party convened in Paris by the European Plant Protection Organization (E. P. P. O.) to look into the possibilities of governmental approach to the problem of seed-borne diseases. Members of the I. S. T. A. and of its Seed-Borne Diseases Committee were present and a full and free discussion took place. A report on the meeting will be published within two or three months but this Conference may like to know that the Working Party agreed in principle with the suggestions outlined above. They did, in fact, prepare a preliminary list of 15 seed-borne pathogens of special concern to some countries, and agreed to invite I. S. T. A. to ask its Seed-Borne Diseases Committee to recommend ways and means by which the risk of carriage of these pathogens with seed can be reduced to a minimum. There was also general agreement that health certificates for seeds should continue to be issued by official Plant Protection Services, though acting where possible in liaison with Seed Testing Stations.

Having dealt with the problem generally, may I now mention one or two specific difficulties. Under the F. A. O. International Plant Protection Convention of 1951, contracting governments undertake to adopt the model phytosanitary certificate given in the Convention for all planting material: and by definition planting material includes seeds. The model certificate states that the consignment, or a representative sample of it, was examined and found "to be substantially free from injurious diseases and pests": It also makes provision for additional declarations when, for example, complete freedom from a specific disease is required. Consequently, unless the Convention is amended, contracting countries which would be quite willing to restrict their demands for health certification of seeds to declarations about specific diseases, must also be given a general health certificate if the exporting country adheres to the Convention, for it is thereby legally bound to the model certificate which must not be altered, e. g. by erasing the clause quoted above.

Another problem arises from the custom some importing countries have of putting special requirements on the import permits they issue. This adds considerably to the difficulties of the exporting country, for the requirements may be changed from time to time, or from consignment to consignment; they are not all included in the printed regulations, and they may be quite unknown to officials in the exporting countries. Moreover, where the requirement involves field inspection of the crop from which the seed was taken it obviously cannot be met if the requirement is not known until the order for the seed is received.

Paper: Problems of testing seed for health

by J. R. THOMPSON
(Seed Testing Station, Edinburgh)

My contribution is from the point of view of the seed analyst.

Now that most of the problems of testing for purity and germination have been solved, there has been for some time a feeling among seed analysts that we should try to test for a third quality, health, and thus ensure that the seeds a farmer buys are reasonably free from harmful diseases. The I.S.T.A. allows tests for diseases to be carried out and reported on its analysis certificates, but only on request of the owner of the seed and only for such diseases as may be specified by him - an omnibus test is not allowed. No methods for testing have been prescribed in the International Rules, but the appendix includes a general guide on how to proceed. While the approach of seed analysts to this problem is cautious, there are enthusiasts who seem to suggest that health is the only quality of seed worth measuring, that testing for purity and germination is old-fashioned and simple. To that school of thought I would say that healthy seed is not of much use if it is of the wrong kind, seed may be quite free of pathogens and yet dead, and seed testing seems easy only because experience has organized and systematized it and fostered a tradition of skill and patience. When you pathologists have built a machine as streamlined, efficient, and accurate as a modern seed testing station you will have something to talk about. However, I hope that most of you take a less extreme view, because for this next phase of development we will be dependent on the help of mycologists, bacteriologists, entomologists, and helminthologists - and possibly virologists too.

I should perhaps at this stage make it clear that the health of seeds is not entirely ignored in our present methods of testing. In purity testing, diseases and pests that can be observed within the limits of the routine test are reported, e.g., sclerotia and nematodes. In a germination test, it is not enough for a seed to prove itself alive - it must produce a seedling robust enough to survive in good sowing conditions in soil. This excludes from those counted as germinable, for example, pea seeds seriously affected by marsh spot and trefoil seeds badly infected with Ascochyta.

That of course is not nearly enough, but it should be realised that our purity and germination tests cannot be adapted to give reliable information about most of the seed-borne pathogens - a third form of testing, for health, will be necessary with suitably qualified staff.

While the universal availability of tests for seed-borne diseases would undoubtedly confer certain benefits on the farming community there is always the possibility of undesirable developments. If one firm advertised freedom from a certain disease its rival might advertise freedom from another, and yet neither disease might be of any real importance. No two purity or germination tests on the same sample ever give identical answers; the same would be true of health tests, and those members of the trade who attach an exaggerated importance to small differences would have another one to complain about. Sometimes, however, the difference is large enough to be taken seriously and this may be partly due to the practice in international trade of having several samples from the same bulk tested and selling on the sample that gains the best report. When the buyer takes over the bulk and has a sample of it tested, the result is almost certain to be lower than the figures

quoted by the seller, and then the dispute begins. There is, too, the danger that the whole business of the seed trade might be slowed up if a health test required longer time than a germination test. Admittedly these are not strong objections to the introduction of health testing, but they are aspects of seed testing in general that should not be forgotten.

What would testing seed for health involve? I estimate that something of the order of 300,000 samples of seed, excluding flower seeds, are tested in the United Kingdom every year. If the Seeds Act and Regulations were amended to make health testing compulsory, that would be the magnitude of the task. The actual cost of a purity and germination test on the same sample, as distinct from the fee we charge, is nearly £1. What would a test for pathogens cost? It is impossible to answer that question with any accuracy because we do not know yet what type of test would be used, but perhaps 12s. per sample would be acceptable as an intelligent guess. So we would have to divert from other purposes enough building materials, equipment, and staff to test 300,000 samples per year and the annual bill would be about £180,000.

It may be argued that this sum is insignificant in relation to the value of the gross national agricultural product, but this product has to bear many costs of which the same could be said and as its value is falling the tendency would be to reduce costs in sympathy. It may be that the cost can be recovered either in higher yields or lower seeding rates or both, but his argument does not justify universal health testing; it only justifies testing for health in those cases in which we are satisfied that the cost is so recoverable. Although we know so much about certain seed-borne diseases there are no data available as to the extent to which seeds sold to farmers are actually infected with them. Our first task must therefore be to find out what are the diseases that are prevalent on the seeds that pass through the trade. This is the line we are working on at East Craigs. Of all the seed samples of the crop under consideration that come in for routine testing, about one in twenty, taken at random throughout the season, is tested by the pathology staff for the presence of pathogens. This shows us which pathogens occur most frequently in the seeds that are being sold to farmers and also the degree to which samples are infected. A survey of oats has been completed and surveys of wheat and barley are in progress. Surveys of this kind will eventually show us which pathogens are worth legislating against and which can be ignored.

Having decided what diseases to test for, the next task is to devise suitable tests. It is not sufficient for the research worker to carry out a few tests and then publish the results. To be acceptable a test must satisfy four requirements:-

1. It must be suitable for routine working in terms of thousands of samples per month.

2. It must be so precisely described and standardized as to be repeatable within reasonable statistical limits in any station and in any country. These "reasonable statistical limits" would probably require at least 400 seeds to be used in each test.

3. Except for supervision, it must not require graduate staff.

4. The answer must be in terms of the percentage of seeds in the sample that carry the pathogen in a viable state. This may not be necessary for quarantine purposes, but I.S.T.A. would insist on it.

How many of the tests already known would, without further investigation, qualify for acceptance under these four heads?

A final point - some seed-borne diseases are adequately controlled by disinfection. In such cases would not a test for adequacy of disinfection be simpler than, and just as valuable as, a test for specific diseases?

There may therefore be good reason for the seed analysts caution. He appreciates the mistakes that were made in the early days of testing for purity and germination, how stations developed their own methods without large-scale statistically-controlled preliminary trials and without reference to other stations and how difficult is the task of disentangling the international chaos that followed. He will therefore insist that before we embark on testing for disease, the preliminary investigational work must be adequate, and unless more specialists are made available for this work it will take a long time.

Paper: Internal seed-borne diseases - a note on their control

by I. A. M. CRUIKSHANK
(Department of Scientific and Industrial Research, New Zealand)

The speaker emphasized that his remarks refer solely to domestic seed trade and not to international regulations on quarantine.

Surveys of the incidence of Ascochyta spp. in lines of resowing seed have been studied over a period of four seasons in New Zealand. The fungi, Mycosphaerella pinodes and Ascochyta pisi, were the most commonly isolated; A. pinodella in very few cases.

The studies indicated distinct seasonal fluctuations in seed-borne infection, which could be closely associated with the growing season, particularly with weather conditions the month before and during harvest. Seed surveys to determine the incidence of Ascochyta spp. and field surveys to study the incidence and severity of collar-rot in growing crops sown from known seed stocks have also been carried out. In all instances crop rotations were of three to four years at least between pea crops; in many instances the fields had been sown down as pasture for ten years.

The data obtained was too preliminary to report in detail. In general seed samples from severely diseased crops yielded up to 20 per cent. infected seed. On the other hand seed lines showing 12 to 15 per cent. infected seed on the basis of the agar-plate test did not necessarily produce severely diseased crops. Similarly seed lines showing less than 1 per cent. on the basis of the agar-plate test did not necessarily produce disease-free crops. The variation in field incidence and severity of collar-rot varied greatly even when the same seed stocks were used on different farms. Seasonal and local factors were probably responsible for a major proportion of this variation.

With these survey results in mind it was suggested that plant disease legislation against seed-borne diseases should not be of the perfectionist type, which might lead to abuse of seed certification in general by the seed trade and farmer alike.

The speaker suggested that a concept, put forward some years ago in Canada, of the establishment of zero tolerance levels of seed-borne infection would be more useful than one based on the presence or absence of disease in a seed line. The zero tolerance level is defined as the minimum level of seed-borne disease, which in an average season for a given growing area could be reasonably expected to yield a crop in which disease would not be a limiting factor. This level of tolerance would naturally be expected to vary depending on the specific disease to be controlled.

Discussion: PROFESSOR MUSKETT (United Kingdom) said that care must be exercised in planning plant disease legislation. He instanced the Destructive Insects and Pests Act, 1907, which stipulated that gooseberry bushes affected by american mildew must be uprooted and burnt, a policy which was followed for some 15 years, until it was shown that the disease could be readily controlled by simple sprays, making legislation unnecessary. He cited also the case of Spongospora subterranea which had crept into nearly every country's list of prohibited parasites, but was in fact unable to survive in many of these territories.

In considering legislative measures for the control of plant diseases care should be exercised to ascertain first of all whether such measures were necessary. He gave two reasons why the problems of seed-borne diseases had now assumed prominence. Firstly in recent years the knowledge of seed-borne diseases had extended considerably and secondly the rising costs in agriculture had made the use of high quality seed imperative.

Although the methods developed in Northern Ireland for the detection of seed-borne pathogens would assist in the formulation of legislative measures they had not been devised for that purpose. On the contrary the work on flax seed had shown that some of the major pathogens of this crop did not affect seed grown in the East and South-east of England, but were only of economic importance in seed produced in the wetter and cooler regions of the West. This indicated that the choice of a suitable area could ensure the production of disease-free seeds and it should be possible by this means to clear valuable stocks of seeds from parasitic contamination.

As examples of areas to be avoided for the production of flax seed he mentioned, Sweden, where Alternaria linicola thrives; Denmark, for which Polyspora lini seems to have a special affinity; and coastal areas, which favour Botrytis.

He urged the prosecution of more intensive research into the etiology of seed-borne diseases before rushing into hasty legislation and he believed that it was the duty of each country to investigate carefully its own problems, with the object of discovering the best methods of dealing with those crop diseases with which it was specially concerned. Knowledge so gained would probably achieve more in the control of seed-borne diseases than purely legislative measures.

The Internation Seed Testing Association Seed-Borne Diseases Committee had already discussed methods for determining the health of seeds and it was hoped that referee samples would shortly be available for distribution to various seed-testing centres. He hoped that suitable methods would before long be available for a wider range of crops, when the question of testing for health would assume a greater prominence in routine testing, and would be carried out by operators trained for that particular purpose.

DR. HANNA (Canada) said that it was well known that plant disease organisms have been and are still being disseminated from place to place by means of contaminated seed. Dissemination may occur within a country as well as from one country to another. From the viewpoint of the grower it was important that he be assured that the seed which he purchased would produce a crop reasonably free from seed-borne diseases; from the viewpoint of the importing country it was important that serious pathogens which did not occur in the country should not be introduced by way of contaminated seed. It was exceedingly difficult to devise regulations that were effective and, at the same time, practicable - regulations that would guarantee freedom from disease and yet not unduly restrict trade between countries. A certificate of freedom from disease was of doubtful value if knowledge of the occurrence and distribution of disease organisms in the country of origin was inadequate, or if laboratory facilities for the proper examination of seed were not available. In some instances it was possible to take advantage of knowledge regarding the distribution of diseases. If it was known that an important disease did not occur in a particular country or region a health certificate based on such knowledge might be more trustworthy than the results of a laboratory examination. For example, Pseudomonas pisi did not occur in certain dry areas in British Columbia, and a certificate to the effect that seed was produced in those areas was a better guarantee of freedom from bacterial blight than the results of laboratory tests. British seed firms had arranged to have some of their stocks multiplied under contract in British Columbia and had agreed to waive the usual requirement of an inspection certificate. Thus the purchaser of the seed was assured of a product free from bacterial blight, and the importing country, was protected against the introduction of a plant pathogen that was not known to occur there. One limitation of the laboratory test was that it could not be regarded as an absolute guarantee of freedom from disease. This fact was recognized in our quarantine regulations which prohibited the importation of seed from regions where certain important seed-borne pathogens were known to occur.

DR. WATERSTON (Nigeria) mentioned some of the difficulties in maintaining an effective plant quarantine service in an under-developed continental territory the size of Nigeria. The long frontier of over 2,000 miles, provided many avenues of entry for plant pathogens. Fortunately as far as seed importations were concerned there was little in the way of private introductions and most consignments arrived through official channels. A plant interchange schedule was maintained between the four British West African territories and good liaison was effected through the West African Interterritorial Secretariat at Accra, which also maintained close contact with French territories.

All seed entering Nigeria was liable to inspection except seed of vegetables and ornamental plants from temperate countries. Steps were being taken to have tomato seed excluded except under permit. A greater use of the permit system for such importation would be welcomed if seed treatment could be imposed before export and again on importation. There was a need to pool all the available knowledge on the distribution of seed-borne diseases and information on the most suitable seed treatments at present known for their control. In this connexion Dr. Waterston wished to re-iterate the plea made by Mr. Moore at the previous conference, for the preparation of an up-to-date world list of seed-borne diseases (including viruses and nematodes) together with a summary of the seed-treatments appropriate for their control.

DR. NATTRASS (Kenya) also emphasized the difficulties of maintaining

a plant quarantine service in an under-developed country, where inspection was frequently carried out by staff not adequately trained for the purpose. Particularly did this apply to small packets of seed which easily escaped detection.

MR. JOHNSTON (Malaya) described the difficulties of intercepting small quantities of seed sent by post and large quantities from China in junks. Those which were intercepted by the customs officials were referred to the plant pathology department, but under present conditions of small staffs no effective control of importation was possible. Seed imported from reputable firms in small packets was automatically passed.

DR. LEE LING (Food and Agriculture Organization of United Nations) stated that the International Plant Protection Convention contained no provision requiring governments to adopt general restrictions on the importation of all kinds of seeds. On the contrary, Article VI, paragraph 2 (a) of the Convention made the matter optional unless deemed necessary by biological reasons.

MR. MOORE, summing up, said that there appeared to be a good deal of support from the Conference for the suggestion that more research, particularly by means of disease surveys, was required to find areas suitable for raising healthy seed. Legislation was necessary but it should be workable, and helpful and this could be facilitated by the expansion of health-testing facilities in seed-testing laboratories and the development of techniques for accurate and rapid determination of pathogens. A distinction must be drawn between the international Orange Certificates and the Phyto-sanitary Certificates necessary for domestic seed-testing schemes.

In answer to Dr. Waterston he said that a list of seed-borne diseases being prepared by Dr. de Tempe and Dr. Mary Noble, in Scotland, and a manual of Seed Tests by Dr. Neergaard, of Denmark, were in an advanced stage of preparation.

DR. WILTSHIRE (C. M. I.) suggested that a resolution should be framed to emphasize the need for further research on laboratory technique and surveys of suitable areas for seed production.

SIR GEOFFREY CLAY stressed the need for research to obtain more detailed information.

It was agreed to refer the subject to the Resolutions Committee (See Resolution 2).

Meeting: Friday, 16th July, 2.30 p.m.

Chair: DR. BATES

Subject: Losses caused by plant diseases in the Commonwealth

Paper: Losses caused by plant diseases in the Colonies

by G. WATTS PADWICK
(Imperial Chemical Industries)

The estimation of losses caused by plant diseases is difficult even in

those countries which have moderately good statistics and services. It is much more difficult where statistics are rudimentary, the peasant farmers have only small areas and are not educated, and the number of research workers for the crop area is low. Dr. Hall of the Commonwealth Institute of Entomology recently said that even after making specific enquiries of the Departments of Agriculture in all the more important colonial territories, the Agricultural Adviser to the Colonial Office was unable to obtain a clear picture of the losses actually sustained "and certainly not of what the losses amount to in the aggregate".

Hall pointed out that George Ordish in his book entitled "Untaken Harvest" makes no mention of crop losses due to pests and diseases in the colonies. It is my intention to try to illustrate the different kinds of losses that occur, depending upon the kind of host plant and the nature of the disease; to show the losses in terms of the industry as a whole; to show that there is a common pattern in disease development even though the precise way in which it is manifested undergoes a variety of modifications; and to illustrate that plant disease losses are progressive rather than static. Finally, because it is necessary to have one common form of expression if a summation is to be made, an estimate will be made of the crop losses, in terms of percentage, for range of crops and groups of crops in the colonies.

THE NATURE AND EXTENT OF LOSSES IN DIFFERENT KINDS OF CROPS

The plant diseases may be divided into those affecting permanent or semi-permanent crops, others attacking vegetatively propagated plants, and diseases affecting crops grown from seed.

(a) Permanent and semi-permanent crops

Permanent and semi-permanent crops are of great economic importance in the colonies.

Three classes of disease in these crops will be discussed, namely, those which kill the host plants, those which have a debilitating effect, and those which cause a loss of crop without necessarily doing permanent damage.

(i) Diseases which kill the trees

Many plantation crops are difficult or costly to establish. If trees are killed in large numbers the loss becomes virtually a loss of capital. I will describe some typical examples.

Swollen shoot of cacao. In 1937, the first mention was made in an Annual Report of the Gold Coast Department of Agriculture of "swollen shoot" of cacao. It was found in the Efiduase area of the Eastern Province in September, 1936. A rapid survey showed about 3,000 acres to be affected in the zone. Within ten years of its detection the disease had spread throughout the cacao-growing areas of the Gold Coast and beyond. It had probably begun on its course at least three years before it was detected.

The officers of the West African Cacao Research Institute have shown that the rate of spread of the disease from tree to tree within an infected area is dependent upon the initial intensity of infection in the area, and they have shown how infection spread over the years within an $8\frac{1}{4}$ acre farm;

between 1945 and 1950, the yield dropped by 86.3 per cent.

Several authorities estimate the number of trees killed to be at least 50 million in the Eastern Province and the quantity of cocoa probably 80,000 tons in 1951. At the up-country price of £3.10.0 a load of 60 lbs. in 1951 this would be worth £10.5 million. In Ordish's terminology, it represents 66,000 acres of "untaken harvest".

The proportion of the world cocoa crop lost through swollen shoot is about 10 per cent.

In industry the cacao trees would be regarded as capital. The Gold Coast Government is prepared to pay 10s. spread over three years for each large cacao tree removed and replanted. This is a very conservative estimate of the capital value, but for all the trees so far destroyed by the disease it represents a capital depreciation of £25 million.

We must recognise that none of these estimates of the loss is wholly satisfactory. Planting in other areas in the Gold Coast has maintained the crop at a slightly higher level than it was ten years ago, but the rural industry of the Eastern Province has been upset and the cutting out programme has had serious political repercussions.

Sudden death of cloves. The disease known as sudden death of cloves appeared in Zanzibar Protectorate at the end of the 19th century. It is characterized by the rapid wilting and death of trees which a few days previously appeared healthy.

In recent years the disease has spread very rapidly and in Zanzibar Island it is estimated that by 1948 half the clove trees had died in the previous 10 or 12 years, leaving about $\frac{3}{4}$ million. In Pemba Island the epiphytotic is at an earlier stage of development. Nutman and his co-workers (1951) who have carefully studied the outbreaks at a number of places found that, as with swollen shoot of cacao, there is a high correlation between the initial size of the outbreak (x) and the annual death-rate (y), the regression equation being $y = 3.49 + 0.327x$. As the average number of trees involved in an outbreak was about 25, the annual death-rate was 11 to 12 trees per outbreak. The average number of trees in an outbreak tended to remain constant because new outbreaks were steadily occurring and older ones eventually ceased to spread or involved the whole plantation. The annual increase in number of outbreaks varied from 11 to 14 per cent., and it was estimated that somewhere between 1965 and 1971 the number of trees in Pemba will have fallen to half what they were in 1930.

The production of cloves in Zanzibar has declined in recent years but has been more than made good by increases from Pemba. This does not benefit the growers in Zanzibar Island, who would appear to have lost nearly 3 million lb. of cloves annually since 1946, worth £840,000 at the 1952 price. The loss in the past six years on this basis is 11 per cent. for the whole Protectorate, and has only been partially counterbalanced by other crops, owing to the slow rate and inadequacy of rehabilitation.

Panama disease of bananas. Panama disease of bananas, caused by a soil-borne fungus, was first reported in Jamaica in 1911-12. From a very early stage diseased trees were required to be removed, and an accurate record was kept through the years of the number infected.

By 1935, 33,000 acres had been lost as a result of the disease, and by 1940, when treatment was abandoned over large areas, more than 40,000 acres must have been destroyed. In 1930 the exports had been over 24 million bunches of bananas but by 1949 $6\frac{1}{2}$ million bunches only were exported. Much of the reduction was a result of the war-time collapse of export, but the recovery was slow. Had prices in 1949 been only half what they were, and had the crop been equal to that of 1930, the value of bananas exported would have been twice what it was, i. e. £$4\frac{1}{2}$ instead of £$2\frac{1}{4}$ million.

Two steps taken have reduced the losses. One is the planting of citrus, coco-nut and other crops; another the introduction of the resistant variety Lacatan. The second solution, however, brought its own problems for Lacatan is particularly susceptible to leaf spot disease, is of lower quality than Gros Michel, and has different temperature requirements in shipping, which raises storage problems.

Panama disease is known in other countries besides Jamaica, and has recently been reported from Uganda.

"Unknown disease" of coco-nuts. Coco-nut palms, which are often planted in Jamaica in areas infested with Panama disease, themselves die from the curiously-named "unknown disease". One thousand acres of coco-nuts were destroyed between 1934 and 1940. During the next decade palms died at an average rate of about 16,000 a year, representing probably 3,500 acres in all. It takes several years for a palm to begin to produce nuts, and it is hardly likely that growing palms to this stage would cost less than £1 per tree, so that during the decade the loss in capital must have been at least £170,000.

It is worth noting that, in contrast to swollen shoot of cacao, sudden death of cloves, and Panama disease of banana, the so-called "unknown" disease of palms seems to have killed roughly the same number of trees each year, and not increasing numbers.

Cypress canker. The infectious diseases hitherto discussed have shown the characteristic of slow but continuous spread over a large area. A more rapidly spreading disease is cypress canker, first found in Kenya in 1946. By 1949 the disease was serious in many places east of the Rift Valley; by 1950 it had spread west of the Rift Valley and was threatening stands on Mount Kenya. The following year it reached Uganda. The effect of the disease, even if it does not kill the trees, is to ruin the timber. In July, 1951, it was announced that no more Cupressus macrocarpa trees would be sown or planted by the Kenya Forest Department and the soft-wood industry in Kenya has now to rely upon other species.

Wilt disease of oil palms. Fusarium wilt disease of oil palms was reported from Nigeria in 1946, and Wardlaw in 1948 found many trees killed. Should this disease follow the same course as the related Fusarium wilt of bananas and spread through native palmeries, the effects on Nigeria's immensely valuable palm-oil industry and on the world's vegetable oil supplies will be disastrous.

(ii) Debilitating diseases

The diseases discussed so far kill the trees sooner or later. There are however, some diseases of great importance which cause a reduction in vigour resulting in little or no crop being produced, although the trees are not killed.

Witches' broom of cacao. Such a disease is witches' broom of cacao. First discovered in Surinam, it had spread to British Guiana by 1906, to West Trinidad by 1928, to Tobago by 1939, and Grenada by 1948. At first it was thought in Trinidad that the disease would not cause much loss. However, in 1933 on the Government estate at Marper the loss was estimated at 1 per cent., the next year 2 per cent., and two years later 6 per cent., despite costly control measures; on other estates the losses averaged 33 per cent. Swollen shoot of cacao, sudden death of cloves, and Panama disease of bananas demonstrate the phenomenon of logarithmic increase in terms of number of trees infected. By contrast, with witches' broom the logarithmic increase is in terms of intensity of infection. Thus in blocks of 100 trees on 24 estates, the number of witches' brooms counted in 1934 was 19, 297; in 1935 it was 38, 021; and in 1936 it was 62, 192.

The export of cocoa from Trinidad and Tobago fell from 1928 to 1952, due in part to the low prices prevailing during the depression and to the interruption caused by the war, but it is probably not unfair to say that 10,000 tons is being lost each year even with the partial recovery since the war. If this is correct, the value at the prevailing world price of cocoa is £4 million, whilst even at grower's price level it is £1. 3 million.

Leaf spot of bananas. Another instance of a disease which weakens the host and spoils the crop is leaf spot of bananas caused by Mycosphaerella musicola. This disease first appeared in Jamaica in 1936, though previously known elsewhere in the Caribbean. Within a year or so it spread throughout the Island and the banana-growing industry could hardly have survived without the help of spraying, which costs about £7 per acre, or a little over £280, 000 for half the bananas in the Island. To this must be added the damage caused by the disease where the trees are not sprayed.

Coffee rust and leaf fall. Since the end of the last century, when rust destroyed the coffee plantations of Ceylon this disease has been recognised as an important factor in coffee production. It is difficult to estimate how much damage is caused for the disease varies in incidence from district to district according to altitude, temperature and humidity, and other factors.

In Kenya, Rayner has found that sprays reduced leaf fall by percentages ranging from 19 to 40. The effect of spraying on yield, which would be the best way of estimating the damage caused by leaf diseases, has still to be determined by experiments over a series of years, but it has been done in India, where coffeee sprayed annually for 20 years has been found to yield approximately double the unsprayed.

(iii) Immediate loss of crop.

It is possible for a disease to take a severe toll of the crop without permanently affecting the vigour of the trees.

Black pod of cacao. Black pod of cacao is widely distributed and is responsible for heavy losses. It appears first as black blotches on the pods and develops so as to render the beans worthless.

Hale estimates the loss in yield of cocoa due to this disease to be 50,000 tons per annum. Thorold (1953) reported experiments in which 40 acres of cacao sprayed to control the disease yielded 10. 2 tons of dry cocoa compared with an estimated yield of 4. 1 tons on an equivalent area without spraying.

We may say then that the annual losses due to this disease are £6.6 million a year, at up-country prices.

(b) Vegetatively propagated crops

Multiplication of crops vegetatively renders them especially vulnerable to the carry-over of disease from season to season.

Cassava mosaic. Cassava, an important food crop in tropical Africa, is widely infected by the virus disease mosaic, which sharply reduces yield. Returns sent to the Colonial Insecticides, Fungicides and Herbicides Committee from plant pathologists in all colonies in which this crop is important show that out of $1\frac{3}{4}$ million hectares devoted to cassava, the equivalent of 200,000 hectares is lost annually due to the disease, which is equal to 11 per cent. loss in yield. A number of resistant varieties have been developed and in time the disease should be brought under control.

(c) Crops grown from seed

Crops grown from seed often suffer from diseases caused by air-borne fungi, which vary greatly from year to year according to weather but the variation tends to be less marked in the case of seed-borne diseases. Some soil-borne diseases spread slowly like the cumulative diseases which kill trees.

Maize rust. In 1949 a rust disease of maize caused by Puccinia polysora, hitherto unknown outside Central America was found in epiphytotic proportions in Sierra Leone. By 1952 it had spread to other parts of West Africa including the Gold Coast, Nigeria, and the Cameroons, and it had also reached Kenya, Uganda, Tanganyika, and Zanzibar in British East Africa. By 1953 it reached Nyasaland and Southern Rhodesia. Rhind (1954) has estimated that in West Africa, where the disease has had the most serious effect, the loss of grain in 1951 was 40 per cent. A trial in which the disease was controlled by dusting in sulphur in 1952 at Ibadan confirmed this figure.

Smuts of sorghum. Grain smut (Sphacelotheca sorghi) is an important disease of the most widely grown of all cereals in the colonies - sorghum. It is seed-borne, and although it does not destroy every grain in the head its incidence is often high and it is responsible for heavy losses. Long smut, too, (Tolyposporium ehrenbergii) is common in some countries. Estimates made in the survey carried out by the Colonial Insecticides, Fungicides and Herbicides Committee already referred to show that the loss in yield due to grain smut alone in the colonies is nearly 7 per cent.

Bacterial blight of cotton. Bacterial blight of cotton is found in most countries where the crop is grown. In its most harmful form, known as "blackarm", the main stem of the plant is attacked by the bacteria (Xanthomonas malvacearum) which cause cankerous lesions greatly stunting growth and sometimes killing the plant.

Research in Uganda has not only shown the extent of the losses but has also led to control measures. In Teso district in 1929, when the disease was severe, the average yield of five improved varieties at three centres where the disease was serious was only 68 lbs. per acre, whereas at two other centres where infection was milder the yield averaged 289 lbs. The yield for the whole country fell from the average of 169,000 bales in the

previous five years to 120,000 bales. In 1933 Hansford established three important facts. He showed that the early stages of infection can be largely controlled by seed dressing, that infection spreads rapidly, and that loss in yield depends largely on the severity of damage to the structure of the cotton plant. The logical step was to extend seed treatment over a wide area, but this was not done until 13 years later, when the Department of Agriculture in conjunction with Imperial Chemical Industries Ltd. undertook extensive trials at Serere. Pottie (1953) stated that in 1950-51 the cotton yield in Teso District where the seed had been dressed was almost double the average for the previous 27 years. It was calculated that the credit for this was shared about equally between the new variety S47 and seed dressing. At the price of cotton lint then prevailing, this additional return was equivalent to a sum of over £1 million. This increase was obtained from 32,000 hectares. The cotton crop of the country is about $\frac{1}{2}$ million hectares.

A study of Uganda Department of Agriculture reports shows that the severity of infection is not everywhere so great as in the district of Teso. If half the increase in yield referred to represents the gain from controlling the disease, and if it operates over half the crop area, the increase obtainable from seed dressing is 25 per cent. of the Uganda cotton crop. If the annual production without treatment is 300,000 bales, the loss is 75,000 bales, which is worth £7 million at current export prices or £3 million to the growers. In fact, much of the cotton seed is now treated at small cost, and the losses are reduced.

Rosette disease of groundnuts. Rosette is a major disease of groundnuts. It exists in several forms according to the strain of the virus, the chief symptoms being mottling, stunting, and distortion of the leaves, with a shortening of the internodes, resulting in a rosette-like habit.

Rosette illustrates the principle that the effect of a disease on yield in an annual crop depends largely upon the stage at which the plant is attacked.

LOSSES IN YIELD OF SOME MAJOR CROPS

This review of some important plant diseases in the colonies should serve to show that the losses caused tend to undergo a continuous process of change - often a steady increase until in a particular area it becomes unprofitable to grow the crop. Therefore, to understand the total effects of the diseases upon the agricultural economy requires a very full understanding of the nature of the disease. Nevertheless, if a summation is to be made of the losses caused by plant diseases in the colonies, a common denominator must be used. The most useful one seems to be the percentage loss in yield.

To this end, estimates have been made which are based on information provided by plant pathologists in the colonies at the request of the Colonial Insecticides, Fungicides and Herbicides Committee. As the information provided was incomplete, it has been supplemented by reference to numerous reports of Departments of Agriculture, by correspondence and consultation, and by personal experience.

The Colonial Plant Disease Survey classifies diseases in three categories, A, B, and C. Class A represents diseases causing over 10 per cent. loss. In this category, the loss can of course vary from 11 to 100 per cent. It has been estimated at the conservative figure of 15 per cent., which may sometimes be too low. Class B is defined as those diseases causing important damage under 10 per cent. and is taken for purposes of calculation as

5 per cent. Class C diseases are those which cause small or very small damage, assumed to average 1 per cent.

In computing the losses, they are estimated for each crop in each territory on the basis of area, so that due weight is given to the important producing countries. Percentage losses are not simply added together, as this introduces the absurd statistical error pointed out by Ordish (1952), namely, an exaggeration of the losses to the extent that they can exceed 100 per cent. A mathematical correction has been made for this error.

Suppose that a disease-free crop is taken as yielding 100 per cent. If a number (n) of diseases causes losses of X_1, X_2, X_3.....X_n per cent. respectively, and if there is no interaction between them when they are operating together, the loss is expressed as:-

$$100 \times \left[\frac{1 - (100-X_1)\,(100-X_2)\ldots\ldots\ldots\ldots(100-X_n)}{100^n} \right]$$

If, for instance, we have two diseases in Class A (15 per cent.) and three diseases in Class B (5 per cent.) the percentage loss is:-

$$100 \times \left[\frac{1 - (100-15)^2 \times (100-5)^3}{100^5} \right] \text{ or 38 per cent.}$$

Where diseases in Class C are concerned, the error is negligible, and therefore corrections are only made when more than one disease occurs in Class A or B.

The estimated losses caused by disease in the various crops are given below, the number of crops concerned being 41.

TABLE I

Losses caused by plant diseases in the major groups of crops and in all crops in the colonies, expressed as equivalent area lost and as percentage loss

Group	Hectares	Percentage loss
Cereals	11,054,224	9.8
Sugar & starch	3,787,250	10.5
Pulses	255,100	5.4
Oilseeds	2,238,100	6.8
Fruits	721,000	5.1
Stimulants, drugs, & spices	1,756,500	25.9
Fibres & latex	3,261,000	18.0
TOTAL, ALL CROPS	23,073,874	11.8

It will be appreciated that reduction in yield hardly suffices to give expression to all the serious losses caused by plant diseases. It ignores

loss in quality and the cost of the control measures adopted. It also fails to take into account disturbances in the agricultural and ecological systems which occur when large areas of trees such as cacao, cloves, bananas, or cypress are killed out. It ignores the social, economic, and even politcal upheavals which we have seen occur in recent years.

Diseases have from time to time affected the kinds and varieties of crops grown, and even the system of agriculture. It is well known that leaf rust eliminated coffee as a crop in Ceylon. It is not so well known that another disease eliminated arabica coffee in British Guiana, and that in several countries this species has been driven out of cultivation and replaced by inferior sorts, namely robusta and liberica. Witches' broom caused a collapse of cacao-growing in British Guiana. It is impossible to say in how many countries virus diseases have prevented the establishment of citrus.

Is it not likely that through the years there has been a natural or deliberate selection of types resistant to disease at the expense of other qualities, including capacity to yield heavily? Perhaps this phenomenon is the rule rather than the exception in colonial agriculture.

Possibly because of rapid methods of transport, there have been spectacular introductions of diseases into the colonies during recent years. Examples are blister blight of tea in Malaya, maize rust throughout Colonial Africa, Panama disease of banana in Uganda, ratoon stunting of sugar-cane in East Africa, and leaf scald of sugar-cane in British Guiana.

The number of plant pathologists is insufficient to deal with the existing problems, let alone to prevent the introduction of new diseases. An increase in the staff of pathologists is essential if the welfare of the agriculturally based industries on which much of our own foodstuffs depends is to be safeguarded for another generation. Likewise, if the colonial peoples with their rapidly increasing populations are to be properly fed, the diseases of cereals and other food crops must be prevented. Neither of these things is possible with the present inadequate staff of specialists.

Paper: Losses from Plant Diseases in Canada

by I. L. CONNERS
(Division of Botany and Plant Pathology, Ottawa).
(Presented by G. B. Sanford, Canada)

Any reliable estimate of losses caused by plant diseases must be based on an accurate estimate of disease intensity. Further, the relationship of disease intensity to the reduction in yield and quality of the crop must be known. In order to establish the relationship between loss and disease several years of careful experimentation is usually required. In this paper, loss estimates obtained in Canada for a few major diseases will be reviewed with only passing reference to the methods used.

For the cereal rusts, the modified Cobb scale has proved excellent for estimating rust percentages. Bailey and Geaney (1925) demonstrated that as the stem and leaf rusts [Puccinia graminis and P. triticina] of wheat were readily controlled by dusting the crop with sulphur loss caused by rust could be estimated. Goulden and Greaney (1930) showed that uniform increases in stem rust resulted in uniform reduction in yield, but that the reduction though uniform was not always of the same magnitude under different conditions.

Using the results of such experiments Greaney (1936) estimated that the loss in yield from rust varied in Manitoba and Saskatchewan from 112,000,000 bushels of wheat in 1925 to nil in 1933. For the 11-year period of 1925-1935, the average annual loss was 35,000,000 bushels. When loss in quality was also considered Greaney concluded that the average annual loss caused by wheat stem rust was almost $33,000,000. By 1938 the new rust-resistant varieties were replacing rapidly the older susceptible varieties except in experimental plantings and the losses caused by rust could be estimated by comparing the yields of susceptible and resistant varieties. Using this method Craigie (1944) concluded that the growing of rust resistant varieties in Manitoba and eastern Saskatchewan from 1938 to 1946 increased the average annual production in the rust area by 41,390,000 bushels of wheat and farm income by $27,242,000. Unfortunately stem rust has again become heavy on wheat since race 15B became prevalent. In 1953 stem rust caused an estimated loss of 25,000,000 bushels of wheat in the rust area in Western Canada.

Losses caused by smut fungi are relatively easy to compute provided suitable counts are made in a representative number of fields. It has been shown by Flor et. al. (1932), Semeniuk and Ross (1942), and Compton and Caldwell (1946) that reductions in yield approximate the percentage of heads destroyed. Gussow and Conners (1927) calculated that the average annual loss from smut in Canada for the 4-year period, 1920-1923, was $12,000,000. This figure was based on estimated percentage losses of 1.2 for wheat, 3.0 for barley, 3.4 for oats, and 2.0 for maize. If these percentages are compared with the estimates of Craigie (1939), Cherewick (1953), and with those for limited areas in Canada given in the Canadian Plant Disease Survey (P.D.S., 19, pp. 17-18, 27, pp. 6-7, etc.) it will be noticed that these percentages are substantially of the same order as those of three decades ago.

Losses from common root rot [Helminthosporium sativum and Fusarium spp.] in Western Canada have also been investigated. Machacek (1943) sampled each year 60 fields representative of the various soil zones in Manitoba for the years 1939-1941. According to him the percentage reduction in yield fluctuated from 8.0 to 16.4, and averaged 12.1 for the 3 years. Sallans (1948), however, has presented convincing evidence that the relationship betweeen common root rot and yields is very complex. As a result of a detailed statistical analysis of data on root rot, insect damage, weather, and yields collected in Saskatchewan for the years 1934-1943 he obtained an estimated loss of 5.14 bushels per acre, with the true value lying between 1.55 and 8.73 bushels per acre. Even the lower limit represents a loss of about 10 per cent. of the harvested yields of wheat.

Estimates of losses from diseases in vegetable and fruit crops are extremely scanty. It may be noted that a crop such as apples could hardly be grown in Canada were it not for the protection afforded by the repeated applications of fungicides (and insecticides). Actual losses may be considerable in a year favourable for apple scab [Venturia inaequalis], but consumer preference and other factors make the loss to the grower difficult to calculate. Losses from physiologic disorders have received some attention in the Okanagan Valley, British Colombia. Undoubtedly the discovery that drought spot and corky core of apple are caused by a lack of boron in the soil has had a profound affect on the industry. The immediate benefit from the discovery was that a loss of 100,000 to 125,000 boxes of apples in 1935 (P.D.S., 15, p. 48) was transformed into a saving of 40,000 boxes in 1936, when the boron treatment was introduced, and an annual saving of 500,000 boxes in 1937 (P.D.S., 17, p. 53). Since then deficiencies of other elements

have been detected and corrected.

In Prince Edward Island in the epidemic year 1951 premature defoliation by late blight [Phytophthora infestans] caused an estimated loss of nearly 1,000,000 bushels of potatoes or about 16 per cent. of the crop (P. D. S., 31, pp. 65-67). It has also been shown that for the years 1945-1951 spraying the plots at Charlottetown with Bordeaux almost eliminated tuber rot and increased the marketable yield on the average by 40 per cent., and never less than 20 per cent., over the unsprayed plots. Yet a field survey in 1951 disclosed that over 10 per cent. of the growers applied no fungicides and under 25 per cent. made sufficient applications to ensure disease control.

The above paper covers the more serious attempts to estimate losses from plant diseases in Canada. Probably we are fortunate in the fact that a few major crops bulk so large in our agricultural economy. A large portion of our research programme centres about these crops and in the course of our investigation of their diseases serious attempts have been made to estimate the losses caused by them. Such estimates have proved valuable because, being based on data which were subjected to rigorous analysis, they have brought conviction to the authorities and public alike of the reality and magnitude of the losses. Reliable estimates are also useful to the extension pathologist in his work of demonstrating the value and use of fungicides in the control of plant diseases.

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Paper: Part I. Some savings brought about in New Zealand by application of measures for control of plant diseases

by E. E. CHAMBERLAIN

(Department of Scientific and Industrial Research, New Zealand)

Dr. Chamberlain said that New Zealand was only interested in feeding its own inhabitants and there was no regular system of plant disease survey; therefore he could only discuss those diseases for which estimates had been made.

Dr. Chamberlain in presenting the first part of his paper discussed the savings effected by the control of the following diseases.

BACTERIAL SPOT OF PLUMS (Xanthomonas pruni) is a serious disease in New Zealand, losses of over 40 per cent. having been recorded in some orchards. Introduction of weak Bordeaux sprays controlled the spot on fruits and reduced twig infection, thereby increasing yield. Spraying has become general with an estimated 15 per cent. increase in marketable fruit in the Auckland district, where the annual value of the crop is £20,000; thus the savings are £3,000 per year. They are at least as great in the rest of New Zealand so that total savings exceed £6,000 per year. GREASE SPOT (Pseudomonas passiflora) and BROWN SPOT (Alternaria passiflora) of passion fruit are two serious diseases which almost put this crop out of production, and cause average losses of about 60 to 70 per cent. The introduction of Bordeaux sprays has reduced these to less than 1 per cent. and has allowed the industry to re-establish itself. Savings to the growers exceed £24,000 annually.

TOMATO CANKER (Corynebacterium michiganense), TOMATO SPECK (Pseudomonas punctulans), TOBACCO MOSAIC, and TOMATO STREAK. - Acid extraction of tomato seed was introduced and has now become standard practice. This method eliminates seed-borne infection of the above diseases and has increased the amount of marketable fruit by at least 5 per cent. As the value of the tomato crop exceeds £1,000,000 savings amount to over £50,000 per year. EELWORM and VERTICILLIUM WILT OF TOMATOES were particularly troublesome in Auckland glasshouses. Following an investigation of the problem improved methods of steam disinfection were recommended and soil treatment with chloropicrin and D.D. was introduced. Soil treatment has now become routine practice. Largely as a result of soil treatment the average yield per plant has trebled in Auckland glass-houses during the past eight years. The increased return to growers exceeds £100,000 a year in the Auckland district alone.

Even with our comprehensive spray programmes for apples and pears in New Zealand it is estimated that 4.5 per cent. of the fruit is down-graded because of BLACK SPOT OR SCAB (Venturia inaequalis) and 0.5 per cent. rejected. This represents a loss of £95,000 a year. However, if spraying was not carried out it would not be possible to grow these crops commercially and an industry worth £1,600,000 a year would not exist.

When ONION SMUT (Urocystis cepulae) was discovered in the onion growing district of Marshland in 1938 over 80 acres of onions were already affected. If it had been allowed to develop unchecked it is estimated that those 80 acres would have been abandoned for onion growing, causing a loss of £6,000. It is also estimated that half of the total area of 9,000 acres of onions would have become infected, causing a further loss of £10,000. The total loss would therefore have been £16,000 annually. The formalin drip treatment was introduced and has become standard practice. Excellent control has been achieved with a saving of £16,000 a year. ONION YELLOW DWARF was also discovered in the Marshland onion-growing area in 1938 and was already well established. From experience in other parts of the world this disease, if unchecked, would have reached an annual incidence of at least 30 to 40 per cent. and the value of crop would have been reduced by approximately 25 per cent. or £5,000 a year. By the introduction of control measures onion yellow dwarf has been eliminated from the district, and £5,000 a year saved.

DRY ROT (Phoma lingam) of swedes formerly destroyed from 25 to 75 per cent. of the crop. The average area sown is between 150,000 and 200,000 acres, valued at approximately £40 per acre. This disease was shown to be

carried in the seed which was almost entirely grown in Europe. By growing seed in dry areas in New Zealand, seed free or almost free from infection is produced. Losses from dry rot are now considerably less and the area under swedes has increased, with consequent increase in stock carrying capacity. The benefit to the Dominion considerably exceeds £250,000 annually.

LOOSE AND COVERED SMUTS OF BARLEY (Ustilago tritici and U.hordei) caused considerable yield reduction and lowering of malting quality of barley. The introduction of hot water treatment of nucleus lines followed by certification of seed crops has eliminated these diseases. The value of the crop produced from of 6,000 acres has been increased by approximately 30 per cent., giving a saving of over £40,000 a year. Control of LOOSE AND COVERED SMUT OF WHEAT (Ustilago tritici and Tilletia foetens) by use of hot water treatment and seed dusts, respectively, was introduced and has become standard practice, being applied by the seed companies to all wheat seed; complete control has been achieved. It is estimated that control of these diseases has increased the value of the wheat crop by 10 per cent. and savings exceed £100,000.

A linseed industry was established in New Zealand during the last war and because of the world shortage of linseed oil has been maintained ever since then. However, our crops were threatened by epiphytotics of RUST (Melampsora lini) and in 1947-48 yields were reduced from an average of 7 cwt. to 3 cwt. per acre, a loss to the growers of £85,000. Introduction of resistant varieties has protected growers from further disastrous crop epidemics and given the industry stability, with an annual saving of £130,000.

Before the introduction of diphenyl wrappers and hormone dips, losses from BLUE AND GREEN MOULD AND STEM ROT OF LEMON were 10 per cent. or £7,500 per annum.

Part II. Some losses caused by plant diseases in New Zealand.

BROWN ROT (Sclerotinia fructicola). It is estimated that in an average year at least 10 per cent. of our total stone fruit crop valued at £80,000, is lost through brown rot. A recent survey shows that 15,000 out of 600,000 trees are infected by SILVER LEAF (Stereum purpureum). At a capital value of 30s. per tree the loss is £22,000 and estimated crop loss is £30,000 a year. STONE FRUIT BLAST (Pseudomonas syringae) has destroyed 13,000 trees (all types of stone fruit) up to eight years old. At £25 per 100 trees the direct loss is over £3,000, potential crop loss is £26,000 per annum, and the loss to industry is even greater as many growers are not attempting to re-establish affected orchards.

With the introduction of fruit-setting hormones BOTRYTIS ROT (Botrytis cinerea) has become a major disease of greenhouse tomatoes. In Auckland the estimated loss is 10 per cent. of £16,000 and the estimate for the whole of New Zealand is 5 per cent. or £30,000. Incorporation of a therapeutant with the hormone is being recommended and losses are now decreasing.

The estimated loss from CLUB ROOT OF SWEDES (Plasmodiophora brassicae) is $2\frac{1}{2}$ per cent. or £150,000 and from SOFT ROT (Erwinia aroidea) of Brassicae is 1 per cent. or £120,000.

VIRUS DISEASES OF POTATOES. - Many of our potato varieties are practically 100 per cent. infected with potato virus X and our most important variety Aucklander Short-taf is nearly 100 per cent. infected with X + Y.

YELLOW-LEAF OF PHORMIUM has become so serious that New Zealand has to import nearly half its hard fibre. If it were not for yellow leaf the country could economically produce its own requirements and probably develop a considerable export trade. With cheaper production of Phormium fibre an expansion of the industry to meet requirements would save the Dominion £1, 300, 000 a year and reduce its overseas expenditure by £2, 000, 000.

Paper: Assessment of crop losses

by R.S. VASUDEVA
(Indian Agricultural Research Institute, New Delhi)

I must confess that we have no reliable figures regarding losses caused by plant diseases in India. All the available information is merely a guess-work of experienced plant pathologists.

If we examine information on losses due to individual diseases in a particular crop it would sometimes appear that the growers raised no crop at all.

It is necessary in the first instance to develop a technique to determine losses due to different diseases under different environmental conditions, i.e., on a regional basis, keeping in view the varieties involved. Later the surveys carried out by the field staff should be correlated so that one could read the loss directly when information on disease incidence in a particular locality is made available. We have collected a certain amount of information on the subject for a few diseases and developed suitable techniques after careful experimentation extending for several years in some cases.

For instance, for black rust of wheat (Puccinia graminis) plants showing different intensities of infection were harvested separately and the loss on 100 plants and 1, 000 grain weight basis was determined. It was observed that loss due to black rust in varieties Agra local, N.P. 4 and Pb 518 when rust infection was heavy (75 per cent.) was 55, 42, and 21 per cent. In recent trials N.P. 10 suffered least in yield under heavy rust infection. Regular controls were maintained by dusting with sulphur which kept the plants free from disease.

Similarly in the case of linseed rust loss in yield under heavy infection of rust (75 per cent.) on 1, 000 grain weight basis was 40 to 50 per cent., and on basis of yield of 100 plants it was 11 to 44 per cent.

For sugar-cane smut [Ustilago scitaminea] '3 budded' setts obtained from healthy canes were inoculated before planting and untreated controls were also planted. At the same time a third lot that came from canes carrying internal smut infection were also planted. Loss due to impaired germination was mainly in the plots planted with smutted setts having internal infection. Loss in yield due to smut infection (external) in Co. 312 and Co. 313 was 11 to 60 and 17 to 73 per cent., respectively. The use of smutted seed (internal infection) involved a loss in both varieties of 71 to 74 per cent.

The incidence of virus diseases (severe types) in potatoes in the plains of India, where the major portion of the potato crop is grown, was observed to be 7 to 94 per cent. during surveys from 1941 to 1945. By simply

distributing partially disease-free seed to the growers, yields, which are normally of the order of about 7,200 lbs. per acre, were pushed up by about 40 per cent. I am sure that under a system of seed certification we can almost double the yields. This shows the tremendous loss that the virus diseases are responsible for in the potato crop in India.

Some work to determine losses due to late and early blight [Phytophthora infestans and Alternaria solani] has been carried out on the lines indicated by Mr. Moore.

The tomato crop suffers seriously from virus diseases wherever the crop is grown and all varieties suffer more or less to the same extent. Severe mosaic, streak, leaf curl, mosaic-cum-leaf curl and leaf curl-cum-streak resulted in losses in yield of 11, 54, 84, 73, and 99 per cent.

Losses ascribed to sugar-cane mosaic by different workers vary from 5 to 50 per cent. consisting of reduced cane yield and deterioration of juice, resulting in low recovery of sugar. To examine the loss due to the disease field scale experiments were conducted in different parts of the country for about 15 years. Healthy and mosaic affected crops were grown in adjacent plots, of about 1/20 acre size in several replications. Necessary precautions to eliminate the border effect were taken. Yield records were determined separately and randomized samples from each plot were analysed for extraction, Brix, sucrose purity, and glucose. The results obtained show that Co. 213 and Co. 313 though susceptible to mosaic, are highly tolerant, i.e., even a very high mosaic infection up to 100 per cent. is not capable of reducing the yield by more than 10 to 15 per cent. No significant difference was observed in sucrose, Brix, purity, or glucose content of healthy and mosaic cane juice, showing that there is no deterioration in the quality of the juice of mosaic affected canes in India. Such a high mosaic infection as in the experimental plots is not met with in the absence of active natural transmission of the disease in Northern India and the losses occurring in practice at present are, therefore, much smaller. We are, however, on the look out as a new strain of the mosaic virus may change the picture in years to come.

Paper: Methods of Assessment of Plant Disease Losses

by E. C. LARGE
(Plant Pathology Laboratory, Harpenden)

Our knowledge of losses caused by plant disease in England and Wales is strictly limited, and we at the Plant Pathology Laboratory and throughout the Advisory Service have a profound scepticism for estimates based on mere guesswork and personal opinion.

In 1941 the British Mycological Society appointed a small committee to devise methods of disease measurement that could be used to reinforce the long-established qualitative plant disease survey work in this country by obtaining more quantitative information. In 1950 a new section for the development of this branch of plant pathology was set up at the Plant Pathology Laboratory, where we are working with the advisory service, and in close collaboration with the National Institute of Agricultural Botany, and other research workers.

In 1952 we started a new Journal, Plant Pathology, one of the main purposes of which is to receive and publish contributions on disease and pest surveys, including the estimation of crop losses, and to build up a corpus of knowledge on appropriate techniques. Thus an organization is now in being for tackling these problems on the basis of large numbers of observations from all over the country.

Turning to actual field methods I will confine myself to the subject I know best, and on which we have made the greatest progress - late blight of potatoes [Phytophthora infestans]. During the war we evolved a simple key which gives descriptions of easily recognizable conditions in the crops corresponding to each of a number of stages of percentage decay of foliage. This key is used in all our work on blight forecasting, and we now receive about a thousand blight observations each year from all over England and Wales. With this number of observations we can draw maps showing the dates of outbreak of the disease each year in each of the several climatic regions of the country; and we can also plot the progress of the disease in each region on a time base. After some point in the progress of the disease on the haulm - say the 75 per cent. stage - there is no further increase in the weight of the tubers. Hence we can map the country for the date at which tuber production is stopped by potato blight. If we then draw an average bulking curve for our main crops (obtained by periodic liftings on a large number of crops) we can read off the mean loss corresponding to the date at which the 75 per cent. stage is reached. If it is at the end of August, for example, the loss is about 13 per cent. of the potential crop, and if at mid-September only about 4 per cent.

It is often assumed that if you spray the potatoes you avoid all this loss, but in fact only a part of it is saved, and in trials during the past six years, we have found, moreover, that wheel damage during the spraying causes a loss of 4 or 5 per cent. of the potential crop.

We do not get the same kind of blight epidemic every year, and indeed in the long run it is only in about one year in three that blight causes much loss on the eastern side of the country. Routine spraying in the dry seasons in which blight does not develop may actually cause considerable loss of yield, not only through damage by passage of the spraying machine, but also by phytotoxic effects.

There may be some long-term gain from routine spraying in certain districts, but we are coming to the conclusion that most of the routine spraying now being done in this country is only on the borderline of being economically justified. Hence the great importance of our work on blight forecasting, for it may help us to dispense with spraying in those years in which it is unnecessary.

Perhaps I have time to mention just two of the other pieces of survey work that we have been doing. One concerns choke (Epichloe typhina) in cocksfoot [Dactylis glomerata] seed crops, and the other common scab [Actinomyces scabies] in potatoes.

In our choke survey we had the advantage of being able to obtain approximate assessments of the disease in all the crops entered for inspection; and these assessments were checked by very careful examination of random samples of the crops. Records from over 2,000 crops during the years 1951-3 showed that this disease, which was thought to be causing great

losses, does not, in fact, cause a total loss of more than about 3 per cent. per annum; and we now know how this loss is distributed according to the age of the stand.

In surveying the incidence of common scab in potatoes, we have used a very simple assessment diagram, and in collaboration with the Potato Division of the Ministry of Food, we have obtained accurate reports on a total of some 5,000 crops in the past two years. This work, which is continuing, is enabling us to define those parts of the country in which scab is most severe; to relate the incidence of scab to type of soil, and to place our potato varieties into at least two main groups for susceptibility to scab. These studies of the conditions of incidence of the disease are of far more value than any mere figures for overall loss, but if anybody wants that kind of figure we can say that the proportion of our potato crop comprised of tubers with a quarter or more of the surface covered with scab lesions was about 4 per cent. in the relatively dry season of 1952, and only about 2 per cent. in the relatively wet season of 1953.

Our work on potato blight and common scab is bringing out some rather interesting and unexpected economic realities. We know that the bad blight attacks occur in the wetter seasons - which favour the growth of the potatoes - so that from a national point of view the effect of spraying is to make a bumper crop heavier still. Scab, on the other hand, is most severe in dry seasons when the crop is light, so that any tubers rejected on account of scab tend to make a small crop smaller still.

Discussion: DR. MAGEE (Australia) said that the position in Australia was similar to that in India where no central authority existed for recording losses from plant diseases. Each state of the Commonwealth of Australia was more or less independent, but what he would say about New South Wales applied generally to the other five states, except that New South Wales did maintain a plant disease survey, which prepared an annual statement. It was generally believed that annual losses from crop diseases in that country were of the order of 10 per cent. but might fluctuate from year to year. It was worthy of note, however, that two crops, namely, rice in New South Wales and hops in Tasmania, had no introduced diseases, and they both approximated to the highest level of production in the world.

A problem in the assessment of losses caused by stem rust of wheat [Puccinia graminis] in New South Wales was that an epidemic year in which five or ten million bushels of grain may be lost is also the year in which the overall wheat production of the State is at its highest. The difficulty was to assess what the yield would have been without the disease - a study of production figures could often be very misleading.

Where compulsory disease control measures had been introduced by the government a very accurate assessment of losses had been possible. For instance with sugar-cane the acreage ploughed out or rogued was expressed in precise figures, and similarly the exact number of banana plants eradicated each year because of bunchy top was known. He could not, however, give accurate figures for the cost of control of banana leaf spot [Mycosphaerella musicola] nor what the losses from the disease were to that industry.

DR. WATTS PADWICK, summing up, said that the contributions made emphasized the difficulties there were in obtaining accurate figures for crop losses, but he found it encouraging to hear of the successful attempts being made in some countries. He himself had encountered the same difficulties in trying to arrive at what he called a common denominator. Mr. Large made a nice point in his remarks on potato blight, namely, that if the average be taken over all the crops of a country every year as an indicator then no spraying at all would be done, yet that, of course, would be the wrong conclusion to draw and the wrong advice to give. In fact, the correct method to pursue was to analyse the disease and study it closely until it was found where a costly treatment might still pay. It was the different sets of conditions that needed close analysis.

DR. WATTS PADWICK went on to say that although this might be what the plant pathologist wanted, it did not necessarily provide the kind of information which appealed to the administrator who controlled the funds, and so often the paucity of information was the direct result of insufficiency of staff due to lack of funds. He was impressed by the start which had been made in England on obtaining large numbers of disease records, using very simple methods of recording and he felt that other countries might profitably follow their example. With this in view he would suggest the passing of a resolution stating that the Conference was in favour of speeding up survey work on losses due to plant diseases and standardizing methods of recording.

The meeting agreed that this suggestion should be passed to the Resolutions Committee. (See Resolution 3)

Meeting: Monday, 19th July, 10 a.m.

Chair: DR. MAGEE

Subject: Cereal rusts and their control
(a) Maize rust in West and Central Africa.

Paper: Observations on Puccinia polysora Underw. in West Africa (summary)

by R.H. CAMMACK
(West African Maize Rust Research Unit)

Puccinia polysora, a leaf rust of maize, was not recorded in West Africa before 1949. Prior to that time the only known rust on maize was Puccinia sorghi, which was of general occurrence, but at no time did it seriously affect the maize growing areas.

P. polysora first appeared in Sierra Leone early in 1949 and rapidly spread eastwards along the coast and across Central Africa as far as Kenya, and south-east towards Rhodesia; by 1953 it had reached the islands of Mauritius and Réunion in the Indian Ocean. During its migration it reached epiphytotic proportions in most affected areas and caused great economic losses.

A survey conducted during 1953 in Nigeria and the Gold Coast showed that the rust was most intense in the coastal rain forest areas, and intense

in the northern regions of the two Colonies. Assay plots sited in the Cameroons under British trusteeship showed P. polysora at 4,000 feet, but not at 7,000 feet.

It was found that the yields of maize were affected by the time of planting, early crops giving high yields whereas those of later crops fell sharply. With early plantings the time of onset of the rust was late and became earlier as the season progressed, the spread of the rust being rapid in early and slower in later plantings. The second rust of maize, P. sorghi, is of general occurrence in West Africa, but has never given rise to an epiphytotic.

The distribution of the two rusts show P. polysora to be mainly confined to the tropics, whereas P. sorghi is most prevalent in temperate and sub-tropical maize-growing areas. This distinction is supported by the biological differences between the uredospores of the two rusts, optimum temperature and humidity for germination being considerably higher for P. polysora than for P. sorghi.

Methods of rust assessment were worked out and investigations [which were described at length] showed that Central and South American maize varieties have provided a source of resistance, and from these, varieties agronomically suited to West African conditions are being bred.

Discussion: DR. BATES (Southern Rhodesia) described how Puccinia polysora appeared simultaneously in Southern Rhodesia, Northern Rhodesia, and Nyasaland and probably at the same time in Portuguese East Africa, which rather indicated that there had been a heavy "spore shower" brought in possibly by appropriate meteorological conditions. He thought it would be interesting to relate the movement of P. polysora across Africa with the meteorological conditions prevailing at the time.

Alarmed at the possibility of severe damage being caused by the fungus the appropriate authorities convened a meeting in Southern Rhodesia, which was attended by representatives of West, East, Central, and South African territories. The problem was fully discussed and lines of research agreed upon.

The rust appeared in Southern Rhodesia in March 1953, late in the crop season when the maize was past the susceptible stage, and no serious damage followed. Leaf material received in April from Portuguese East Africa, which had been grown under conditions of higher rainfall and temperature, was very severely affected, suggesting serious losses from the disease; this was confirmed by later reports.

As a result of the meeting held in Salisbury, numerous varieties of maize having possible resistance to P. polysora were sent to Southern Rhodesia by research officers in the affected African territories and were grown in field trials in both the high and low veld together with suspected susceptible varieties. Unfortunately severe drought was experienced and there was less infection by P. polysora than in the first year of its occurrence. Indications are that the disease has been less widespread and less severe in 1954 than in 1953, suggesting that its incidence is governed by climatic conditions, particularly temperature and humidity. Unless a very favourable combination of these two factors occurs it does not seem at the moment that heavy losses will be experienced in the major maize-

growing areas of South-Central Africa. However, as a result of the discovery of the teleuto-stage in S. Rhodesia, and possibly Nyasaland, dangerous physiologic races may appear.

The assessment of losses from the disease in Central Africa is complicated by the late appearance of the fungus, which then usually attacks the top leaves. If it comes before the grain has properly formed this is of importance, as it is the upper leaves which are closely connected with the filling of the cob. In determining losses attempts were made to control the rust by sulphur dusting. This, however, reduced the pH of the soil so much that the growth of the maize was adversely affected. Dithiocarbamates were successfully substituted for sulphur, and the most recent information suggests that on a susceptible variety in Nyasaland the probable loss due to P. polysora is only of the order of 17 per cent.

MR. MOORE (United Kingdom) asked if Mr. Cammack was satisfied of the validity of evidence for the reported spread of the rust from West Africa to Madagascar in so short a time. Citing antirrhinum rust as an example he stated that a perusal of the literature gave an impression of its sudden appearance in southern England, in 1933, with subsequent rapid spread to north-western and southern Europe, when in fact there was strong evidence of its occurrence in England in 1921. The summers of both these years were unusually hot and dry and it was possible that climatic conditions had favoured the disease so that it was readily observed. Once a disease is on record people are inclined to look for it, and new records of its occurrence suggest a rapid spread. Might it not therefore be that P. polysora and P. sorghi have been present as mixed infections in Africa for some time and that favourable climatic conditions inducing unusual development had focussed attention on the former?

MR. CAMMACK said that herbarium material of maize rust in Sierra Leone, Gold Coast, and Nigeria had been closely re-examined by competent mycologists and it is certain that P. polysora did not appear in West Africa before 1949.

DR. MAGEE (Australia) said that antirrhinum rust in Australia had some bearing on the problem of spread, and gave an account of the attempted eradication of the disease after the first report of its presence in October 1952 in Sydney. Despite all efforts with considerable staff the rust reached Victoria within a few months, was reported in Tasmania after a year and in New Zealand in December 1953. There had been no records of the disease prior to this, and the plant disease surveys in Australia were of a high standard. This rapid spread he interpreted as being by wind currents, probably in the upper air.

MR. RHIND (Colonial Dependencies) said that from his experience in West Africa he would like to emphasize that P. polysora is a purely tropical rust, which sets up severe infection only under conditions of high temperature and humidity. However, where rainfall is continuous and exceptionally heavy, spores are apparently washed on to the ground and attacks are frequently less severe.

Regarding the distribution of the rust in Africa he had no doubt it was a new introduction, probably in 1949. It may have first appeared in Liberia somewhat earlier than this, but there were no records to check the possibility. He confirmed that there were no records previous to 1949 in Sierra Leone,

Gold Coast, or Nigeria, where all old records were of P. sorghi and later ones of P. polysora; the same was true of East Africa, Mauritius, and Madagascar. If P. polysora continued its progress across the Indian Ocean, which seemed probable, it would eventually reach the Far East, where it would be likely to cause heavy losses as the maize varieties grown in India, Ceylon, and Malaya were known to be highly susceptible. A similar sequence of events to those in Africa, namely heavy losses in hot humid areas but less destruction in hill areas, might be anticipated until resistant varieties had been established. West Africa was fortunate in possessing highly resistant Mexican varieties for breeding work.

MR. CAMMACK in reply to a question regarding the appearance of new physiologic races of P. polysora and the production of inbred lines of maize, stated that several seed lines had been tested against the rust from many regions of Nigeria and the Gold Coast, and all had reacted uniformly. At the moment therefore only one strain of the rust appeared to be present.

(b) Wheat rust and its control

Paper: Wheat rust in Canada

by T. JOHNSON
(Laboratory of Plant Pathology, Winnipeg)
[Presented by W. F. HANNA, Canada]]

In this paper, the main emphasis was placed on an evaluation of the control measures against wheat rust that have been tried or are now under trial in various parts of the world. Particular reference was made to North America and, as stem rust (Puccinia graminis) is the most destructive of the cereal rusts on that continent and the one against which the major efforts for control have been made, attention was primarily focussed upon it.

Control measures that have been used with greater or less success are chiefly the following:

(1) Destruction of the alternate (aecidial) host.
(2) Chemical control
(3) Restriction of cereal growing in critical locations.
(4) The growing of resistant varieties.

The applicability of control measures, of course, depends upon circumstances. For example, to state the obvious, the destruction of the alternate host is useful only in those regions in which it is of importance in propagating the rust. The use of chemicals (at least, the chemicals thus far tried) is rather too expensive except for crops with a high value per acre. Restriction or prohibition of the growing of cereals is useful only under the special conditions in which a small territory acts as a source of inoculum that can be eliminated by the absence of the host plant. Finally, control by the growing of resistant varieties rests on the assumption that it will always be possible to develop agronomically useful cereals resistant to the rusts prevalent in the region in which they are to be grown, an assumption that should not be too readily taken for granted.

Destruction of the alternate host

Discussing the present status of these control measures, the author said

that North America provides the best example of a large-scale application of this control measure. The reference, here, is to the extensive campaign for the eradication from the Mississippi Valley States and adjacent parts of Canada of the common barberry (Berberis vulgaris), the aecidial host of Puccinia graminis. This plant was introduced from Europe in the seventeenth century into the New England States and became widespread. Eradication was begum in 1918 and up to the end of 1953, more than 448 million bushes and seedlings had been destroyed. The fact that stem rust is still a very important disease in the Mississippi Valley area may raise the question as to whether or not this great expenditure of energy and money was worth while. But if it had not been done the barberry would have become naturalized and innumerable local rust outbreaks would have coalesced to form larger ones, resulting in an earlier general rust infection. Perhaps even more important the barberry would have served as a breeding ground for new physiologic races of the rust. As a result of eradication the barberry performs neither of these functions to any important degree in the central parts of North America. The rust that moves up each summer from Mexico and Texas to the plains of Western Canada comes chiefly from overwintered urediospores, and the race population is undoubtedly much less diverse than it would be if no barberry eradication had been undertaken.

Chemical control

The importance of chemical control of rust lies less in its actual usefulness up to the present than in the promise it may hold for the future if other means of control should fail. It was shown by F. J. Greaney and others, in the years between 1925 and 1935, that infection by cereal rusts could be controlled by sulphur dusting but the method was so laborious and expensive that its usefulness was largely limited to cereal crops of a very high value per acre.

The production of rust-resistant cereals from 1935 onwards served as a deterrent to further investigations on chemical rust control. Conversely, the outbreak, in 1950, of race 15B of wheat stem rust was a great stimulus to studies on chemical control because of the susceptibility to this race of wheat varieties formerly resistant.

In recent studies the search has been for systemic chemicals that would protect the plant against rust infection for a considerable period. Some promising fungicides approach this desideratum. Calcium sulfamate, for example, has a prolonged protective effect and, moreover, has some value as an eradicant of established infection. Unfortunately its systemic action is sometimes reflected in a marked reduction in the viability of the seed produced by the treated plant. Nevertheless, the results of the search for a chemical with the requisite systemic properties are sufficiently promising to indicate that it should be continued. In the future, the ups and downs of other control measures should not be allowed to influence this type of investigation unduly.

Restriction of cereal growing in critical locations

Where a limited area serves as a source of inoculum for large and important grain-growing regions it is theoretically possible to reduce rust losses in the more important area by prohibiting grain growing in the less important one. For political and economic reasons this type of control would frequently be difficult to apply, but some measure of success has been

obtained in India. The late Dr. K.C. Mehta realized many years ago that the Nilgiri and Palni hills served as a source of stem and leaf rust inoculum for the neighbouring plains by permitting the rust to oversummer on spring-planted grain. It appears, from recent reports, that prohibition of the growing of wheat and barley, in summer, in the hills has had the effect of reducing the amount of stem rust in the fall-planted crops in the plains. (Misra, C. P., Ind. J. Agric. Sci., 23, pp. 47-54, 1953.)

It seems possible that a similar beneficial effect might be obtained in North America if it were possible to prohibit the growing of wheat in the stem-rust overwintering areas adjacent to the Rio Grande River (southern Texas and north-eastern Mexico). Since it is improbable that such a scheme could be executed, an alternative one of growing resistant cereals in this region, might minimize the annual northward spread of rust. There is, however, relatively little incentive to the production of rust-resistant winter wheats for the southern United States for the reason that winter wheats generally escape severe rust damage through early maturity. The rust susceptibility of the winter wheats is a matter of great concern for the growers of spring wheat in the northern states and Canada because they serve as a source of inoculum for the much later spring wheat region.

The growing of resistant varieties

The growing of resistant varieties has been the major method of rust control for at least 30 years following the work of Biffen early in this century. He was the first to show that rust resistance is a heritable characteristic conditioned by Mendelian factors or genes.

Later Stakman et al., from 1916 onwards, showed that stem rust is made up of many physiologic races relatively stable in their pathogenic behaviour. Therefore, if a variety can be found that is resistant to the race population of a unit area (such as North America), it will remain resistant unless new races pathogenic to it arise or are introduced. If no such variety can be found it may still be possible to synthesize it by combining resistance elements from two or more varieties.

Obtaining such a resistant variety is only a first step in a breeding programme for rust resistance because it is rare for such a variety to possess the properties of yield, bread-making quality, etc., that are desired by growers in various regions. A period of at least ten years is likely to elapse from the discovery of the source of resistance to the production of the economically useful variety. It is, therefore, very important to ascertain that there are no races present in the region under consideration that are virulent to the source of resistance employed in the breeding programme. Two means are employed to this end. The first is the use of annual physiologic-race surveys embracing rust collections from all parts of the area. These rust collections must not only be submitted to analyses of the physiologic races present in them but must also be tested on the resistant variety under consideration to provide assurance that there are no strains present that are pathogenic to it. The second is by growing the variety in so-called "rust nurseries" judiciously distributed in places where rust is generally prevalent.

Having ascertained that the source of resistance is adequate against the rust, it must then be incorporated in an agronomically useful variety. Until recently, the adequacy of this method of rust control was a matter for

conjecture as the answer could be given only by the performance of the new varieties over a period of years. This is worth a brief consideration as the past must serve as a guide to the future.

The first highly stem-rust resistant varieties of good baking quality produced in North America were Thatcher, released in 1934, with rust resistance chiefly from Triticum durum, Renown, released in 1936, with resistance chiefly from T. dicoccum, and Apex, released in 1937, with resistance from both sources. These and similar varieties were highly resistant to stem rust until 1950 when race 15B suddenly gained wide distribution, although its existence had been known ten years before. Similar experience in Australia and Kenya Colony, where the rust resistance of bread-wheat varieties has had an even shorter lease of life, emphasizes the need for many different rust-resistance sources; for it is obvious that when the plant breeder finds that a given source of resistance is inadequate he must have available another one to meet the new situation. Annual rust surveys permit the plant breeder to produce a useful variety with added resistance before a new race becomes widely prevalent. But this warning is useless if no source of resistance to the new race is available. Hence the need for many and distinct sources of resistance.

When race 15B was discovered about 1940, a search was begun to locate adequate sources of resistance. These were found in Red Egyptian, certain wheats from Kenya Colony, and the Canadian wheat McMurachy, and this led to the production of the 15B-resistant variety Selkirk of which some 150,000 acres were sown in Canada this year. But it is already known that Selkirk, at the beginning of its career, may be rusted by a substrain of race 15B, designated 15B-3 which, fortunately, at the moment, is present in only trace quantities. The Kenya wheats K. 338, AC. 2. E. 2, and K. 117A happen to be highly resistant to this and other substrains of race 15B and are already in use in breeding programmes in Canada, the United States, and Mexico. Commercial wheats with the type of resistance they possess should soon be available, and should suffice against known North American races, but there is no guarantee of a long duration of this kind of resistance because races are known, in East Africa, that can attack both K. 117A and K. 338.

Active search for new sources of resistance within the genus Triticum and the introduction of new resistance genes to wheat from other genera are more than ever necessary. Already wheats with high stem-rust resistance have been produced from crosses with Agropyron elongatum, and crosses with Aegilops, Haynaldia, and Secale are receiving attention.

A bright feature of the present situation is the recent organized international co-operation in the establishment of a number of rust nurseries in Canada, the United States, Mexico, and South America, in which possible sources of resistance to stem rust, leaf rust, and yellow rust are tested against a multiplicity of pathogenic types under a great variety of environmental conditions. Under this scheme a gradual "screening" of more than 13,000 accessions of the World Wheat Collection of the United States Department of Agriculture is being accomplished as a part of the search for rust resistant wheats. For the first time in history a united effort is being made to use Man's knowledge and experimental ingenuity to protect his most valuable food crop against its most destructive diseases.

Paper: The Wheat rust problem in India

by R.S. VASUDEVA
(Indian Agricultural Research Institute, New Delhi)

Wheat, the second most important cereal in the Indian Union, covers an area of about 24,000,000 acres, which yield approximately 172,000,000 maunds so that the yield is as low as 8 mds. (660 lbs.) per acre. The crop suffers from the black, brown, and yellow rusts (Puccinia graminis tritici, P. triticina, and P. glumarum). Of the three, yellow rust is limited in its distribution, being restricted mostly to the northern parts of the country. In the south black rust was most common, but brown rust appears to be assuming greater importance in recent years. No reliable figures for the over-all loss resulting from rust epidemics are available, but it has been experimentally determined that the loss in grain-weight may vary from 10 to 50 per cent. depending on the time and intensity of attack and the wheat variety. The annual loss has been estimated at Rs. 49 million, and in epiphytotic years as in 1947 it is much greater, but it has been shown that the shrivelled grain, except in extreme cases, can be used safely for seed.

In the Indo-Gangetic plain, which is the main wheat growing tract, the crop is sown in October-November and harvested in March-April. Yellow rust appears in January followed by brown and black rusts in February-March depending on the weather. In places that are situated near the foothills, such as at Pusa in Bihar, rust occurs much earlier, brown rust having been observed as early as 9th December. From the time of harvest in April until the end of October there is no host in the plains on which these rusts could oversummer, and the heat is so intense (shade temperatures exceed 110°F.) that the uredospores cannot remain viable for a period of 5 to 6 months.

In the hills on the other hand wheat is sown during September-October and harvested in May-June, depending on the altitude. Consequently there is a gap of only two to three months when there is no standing wheat crop. On account of favourable climatic conditions, uredo- stages of all the three rusts have been found to oversummer at different altitudes in the hills (4,000 to 8,000 ft.) on volunteer plants, stubbles, and tillers, which are responsible for early infection of the normal crop sown early in winter in the hills. This region is therefore independent of Berberis for the perpetuation of black rust.

Out-of-season crops also help in the perpetuation of the rust during the critical period, and in the early dissemination of the inoculum to the regular winter crop. In Central Nepal the prematurely sown crops planted in August-September at altitudes of 4,000 to 5,000 ft. have been found infected with black rust in the first week of December. Early outbreaks of rust have been observed in the foot-hills and plains along the Nepal border. The Nepal range is obviously the most dangerous focus for the crops in the Indo-Gangetic plains, although oversummering is possible all along the Himalayan range at suitable altitudes.

Till recently it was considered that the main foci of infection in South India were Nilgiri and Palni hills and that the suspension of wheat cultivation in these areas would help in keeping check on the ravages of rust in the southern parts of the country. Surveys conducted in Western Ghats have, however, modified this view as oversummering of black and brown rusts is possible in these areas too. In October last self-sown plants infected with black rust were found in Panchgani and the following November black and brown rusts were

present at many places in that area. In the same district a heavily infected plot was found in the last week of November. The abundance of inoculum of black and brown rusts around Panchgani when the crop at lower altitude was practically free of rust is strong evidence that oversummering of black and brown rusts is possible in Western Ghats where the climatic conditions are suitable for survival of uredospores.

Two crops of wheat are cultivated in the Nilgiri and Palni hills and the sowing of wheat in Mysore and Bombay at an elevation of 3,000 ft. commences from July and continues till December. The July-sown crop gets infected in September through uredospores disseminated from the Nilgiri hills and a multiplication of inoculum takes place in the immediate neighbourhood of fields sown subsequently, resulting in epiphytotic conditions in years of favourable rainfall.

Wild grasses have been reported to be collateral hosts of these rusts in several countries, but the little work done in India so far provides no definite evidence of the perpetuation from one season to the next through grass hosts. The occurrence of P. graminis tritici on Bromus patulus and Brachypodium sylvaticum was reported, but the growing period of these coincides with that of wheat. Two grasses, Vulpia myuros and Briza minor, have been reported to be infected with P. graminis tritici from South India, and it is felt that these may carry over the rust during the off-season. At Delhi V. myuros has been successfully infected (resistant type of pustule) with the races of black rust of wheat under glasshouse conditions. Besides these two, eleven other foreign grasses, viz., Bromus coloratus, B. carinatus, B. mollis, Hordeum distichon, H. murinum, H. stenostachys, Agropyron semicostatum, Lolium perenne, Hilaria jamesii, Aegilops squarrosa, and A. ventricosa have been found infected by P. graminis tritici under natural conditions. A few grasses, B. catharticus, B. japonicus, and Hordeum murinum, have been reported to be susceptible to yellow rust. Besides these, a fairly good number of exotic grasses have been found susceptible to more than one rust when tested in the glasshouse. It appears reasonable to assume, therefore, that a few of them like Lolium perenne, a perennial grass in the sub-Himalayan hills, may carry over the rust infection from one crop to the next.

In view of the oversummering of rusts in the hills it is now our policy for all imported grasses to be tested against Indian physiologic races prior to their release for general cultivation. Intensive search for collateral hosts has also been taken up.

In India studies relating to dissemination of rusts are now being carried out at 60 representative stations. The slides exposed at these stations, where rust nurseries are also located, are changed twice a week from November onwar till the date of rust appearance. At certain stations, particularly in the foot-hills of South India, the slides are exposed for six to eight months in a year. This should give some useful information regarding the probable date when the spores reach a particular place and later may be correlated with the wind currents during those periods. It may also indicate the foci where the rusts can oversummer. These studies, however, have their limitations, as the uredospores may be of any physiologic species of P. graminis other than P. graminis tritici, and the dates of appearance of rust recorded by different agencies all over the country cannot always be relied upon.

The Indian Ocean in the south and the vast Himalayan ranges in the north would normally act as reasonably effective barriers for the dissemination of spores from other countries; they could, however, arrive to a limited extent

from Afghanistan, Pakistan, and Burma and the possibility of introduction of new races of rusts from countries situated widely apart, cannot be ruled out with the inception of air communications.

The connexion between Berberis and black rust is well known. In the plains of India, which cover more than 95 per cent. of the area under wheat, there is no Berberis and consequently the aecial stage does not come into the picture and alternate hosts do not appear to play any significant part in the annual recurrence of rust; most of the species of Berberis in the hills are other than B. vulgaris. Some species of Berberis, e.g., B. lycium, present in the hills have been shown to be susceptible to the sporidia of black rust and this aspect requires further investigation. The teleutospores remain viable at high altitudes and there is reason to believe that at such places infection would occur. Further the possibility of the occurrence of B. vulgaris in Nepal and the interior of the sub-Himalayan range cannot altogether be ruled out. These areas are now being explored. A species of Berberis has recently been found infected by rust in "Lahul" valley. The presence of these barberries in the hills may possibly explain the gradual rise in the number of physiologic races in recent years. The appearance of new races cannot, however always be attributed to the possible infection of Berberis but may be the result of mutation. So far no case of mutation involving variation in pathogenicity has, however, been recorded in India.

A study of physiologic races of the rusts is in progress at Simla sub-station of the Indian Agricultural Research Institute on an all-India basis, so that this station serves as the nerve centre of the entire rust work in the country. So far about 150 samples of the three rusts received from different parts of the country are being analysed every year, but considering the acreage covered by wheat and the size of the country this number is inadequate and with increased facilities it is hoped that at least four times the number will be analysed in future. So far 10 races and 2 biotypes of black rust(1), 8 races of brown(2), and 10 of yellow(3) have been recorded in India. Some of these cultures have completed more than 300 generations without exhibiting any change in pathogenicity. Amongst these, races 21, 40, and 42 of black rust, 10, 20, and 63 of brown rust, and 19 and 31 of yellow rust are most frequently met with. It is, however, interesting to note that race 15 of black rust which was a common race some years ago, is not frequently met with now, whereas race 21,rare before 1942,has come to occupy a prominent position. Information available on the factors responsible for the predominance of certain races is, however, very meagre. The climatic conditions prevailing in a particular region of the country and the variety would play an important role in the persistance of a particular race. With the introduction of new and improved wheats some of the unimportant races and others so far unknown may come into prominence, and this aspect is being carefully watched.

In view of the fact that Berberis and Thalictrum, the alternate hosts of black and brown rusts, do not appear to play any important role their destruction will serve no useful purpose. Also the species of Isopyrum reported to be an alternate host of P. triticina in Siberia does not occur in India. No alternate host of yellow rust has been discovered so far. It was suggested

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Footnote: (1) Races 15, 21, 24, 34, 40, 75, 117, 122, 194, 15C, and 42B.
(2) Races 10, 11, 20, 26, 63, 106, 107, and 108.
(3) Races 13, 19, 20, 31, A, D, E, F, G, and H of yellow rust have been identified. The last 6 races of yellow rust have not yet been assigned International numbers.

that since the sources of the annual recurrence of these rusts lie in the hills and cover five per cent. of the total acreage under wheat, epidemics may be controlled to a large extent by the elimination of inoculum from pockets of oversummering wheat by the destruction of volunteer plants, suspension of summer crop in Nilgiri and Palni hills, by replacement of wheat and barley by oats, or by cultivation of rust-resistant varieties at such altitudes. Due to the possibility of oversummering in the Western Ghats and in the absence of knowledge regarding collateral hosts the suspension of wheat cultivation in Nilgiri and Palni hills in the south does not appear to be a practical proposition, neither does the destruction of volunteer plants in the inaccessible areas in the hills. As a measure to control rust in the plains, the cultivation of wheat in the Nilgiri and Palni hills was banned for five years, but it gave no satisfactory results and contrary to expectations a high incidence of rust was reported in the plains during the enforcement of the ban. Control of the disease by the use of fungicides also does not apppear to be a practical proposition so far as our present knowledge goes, in view of the cost involved. Dusting with sulphur to ensure at least the seed supply for the following season was suggested at the time, but the cost has been estimated to be much more than that of the entire wheat produced so that treatment with fungicides over a large area at present is not economically feasible in India.

Cultural practices which favour early maturity of the wheat crop have been shown to lessen the destructiveness of rusts, as they help the crop to escape the most serious effects of the disease, but this again has very limited application.

Undoubtedly the most effective method of control is the cultivation of resistant varieties. It is also the best method to eliminate the inoculum in the pockets where oversummering takes place. This method is, however, arduous and long-drawn out. For northern Indian hills a few varieties have been devoloped and selected and are now being distributed to the growers. Ridley, an Australian variety, is fairly resistant and has desirable agronomic characters, whilst NP 770, a selection from a cross between Japanese wheat Kononso and NP 4, is highly suitable for the hills and it is hoped that these varieties will meet the long felt need. Introduction of resistant varieties to the hills may not be effective in checking rust unless all other regions, such as Nepal and Pakistan, are included. Even then the rusts may perpetuate on grasses and may bring about epidemics but the introduction of resistant varieties in both hills and plains would mitigate the losses.

Breeding work is in progress at the Indian Agricultural Research Institute and in the States. Since all the indigenous varieties of wheat tested were found to be susceptible to Indian races of rusts it was considered necessary to cross them with foreign resistant varieties. W 206 is a cross between the famous 'Thatcher' and NP 165. Some selections of crosses which have now been assigned NP numbers such as 789 and 790 are highly resistant to all the known Indian races of black rust and are finding favour with the wheat breeders. Other important crosses such as W 245 and W 246 are between Kenya wheats and NP 165 and NP 120, respectively. Some selections from Cross W 245, namely NP 797, NP 798, and NP 799 have done very well regarding yield and rust resistance in the trials conducted in different States in the country.

For brown rust resistance some foreign wheats like Mediterranean and Democrat have been crossed with NP 114 and Pb. C. 518, respectively, to combine resistance with good agronomic characters; for yellow rusts foreign

resistant varieties, such as Spalding prolific and Carsten V, have been crossed with NP 114 and Pb. C. 518, respectively.

At the same time attempts are being made to produce a good variety which might be resistant to all the three rusts and their races, and with this objective in view Spaldings prolific and Democrat wheats were crossed with NP 114 and Pb. C. 518, respectively, and their progenies (F8 generation) crossed together. The hybrid so obtained was crossed with Kenya wheat to introduce the resistance for black rust. One selection from this, NP 809, is showing promise in the low elevation areas of the hills. These few crosses which I have mentioned are outstanding, but a very large number of crosses are made and studied every year. The subject has gained greater importance since the partition of the country, resulting in the loss of an important wheat producing belt. Proper study of these problems has been taken up on an all-India basis by a team of plant pathologists, geneticists, and plant breeders.

Discussion: DR. BELL (Cambridge) said that at Cambridge they had synthesised amphidiploids between species of Triticum and Aegilops and other related genera, and also amphidiploids within the species. These had been listed for resistance. The Aegilops involved in the crosses were resistant to races of P. glumarum and P. triticina but on the whole the amphidiploids obtained were not as promising as their parents might suggest. As far as P. glumarum was concerned they had developed very little resistance, though there was hope in two cases. They considered the future more hopeful for P. triticina, although it looked as if the resistance was carried by the recessive factor, which was concealed in the amphidiploid. He did not know much about their resistance to P. graminis, but believed that they had had some promising results for resistance to race 15B with some of those amphidiploids sent to Winnipeg.

DR. HANNA replied that in Canada they were interested in producing resistant varieties, but fortunately they could develop these without becoming involved in very difficult genetic problems. They used material that they knew would give good crops.

MR. SAMUEL pointed out that DR. HANNA had described race 15B, saying that it was known in 1940, but did not develop in epiphytotic proportions until 1950. He wanted to know if there was any evidence that in the intervening years it was gradually working its way down to Mexico and then there was a big build up of spore inoculum which came back in 1950.

DR. HANNA replied that he did not know of any evidence. However, he thought it must certainly have spread down to South Texas and Mexico and overwintered there to cause the wide and sudden outbreak in 1950. He was not satisifed that it had appeared earlier for if it had it was remarkable that the surveys in the United States had not shown it. He went on to say that it must be realized that if a single variety or a number of varieties with common or similar parentage were grown over a very large area it was inviting this type of outbreak. The Marquis crop was general at that time. There was the same problem with oats when developing crown rust resistance. However, this did not explain the sudden outbreak of race 15B.

DR. WILTSHIRE said he was interested in the control of rust by fungicides. DR. HANNA and DR. VASUDEVA had both found sulphur uneconomic. He wondered if calcium sulphamate would be economic. He asked this as it

might have been useful, for instance, in the Puccinia polysora epidemic in the Gold Coast to have known of a fungicide that could have been applied immediately to mitigate the effects of the epidemic even though it was uneconomic for general use. Despite the rapid progress in producing resistant varieties the setting up of the maize rust research unit in West Africa took some time.

DR. HANNA said that he thought in the United States calcium sulphamate had become systemic and affected the grain. Experimental work had been limited, and he did not know about the economic aspect.

MR. CAMMACK said that sulphur had been used at Moor Plantation, Ibadan, to assess the losses from Puccinia polysora in West Africa in 1951 and a partial control of the rust was obtained. T. M. T. D. S. gave a high degree of control but the price was prohibitive in the dressings required and it had a marked phytotoxic effect on fertilization. P. P. mercuric also showed a certain measure of control, but he did not believe it would be economic on a large scale.

DR. BROADFOOT (Canada) said that he had had success with sulphur at St. Paul, in Minnesota, but the cost had not been ascertained.

DR. HANNA said that in Western Canada and the United States chemical manufacturers were interested in the question and research was going on.

DR. VASUDEVA said that in India they could get cheap chemicals, but the cost of the labour and machinery involved was not commensurate with the value of the crop. Something really cheap was necessary. Chemical control would be all right for protecting a crop for experimental purposes but in the present state of knowledge they had no evidence that it would be suitable on a large scale.

Subject: The rapid identification of bacterial plant pathogens

by C. R. MILLIKAN
(Department of Agriculture, Victoria, Australia)

In all cases where one suspects the presence of a new bacterial disease a quick and positive identification of a bacterium is essential to enable the necessary Governmental action to be taken to limit its spread or attempt its eradication, should that be feasible.

However, under the conditions prevailing in the average laboratory, the rapid identification of plant-pathogenic bacteria is not possible; in fact it is a tedious and time-consuming process.

It involves inoculation tests to prove pathogenicity, biochemical tests which may take several weeks, and serological or phage tests. Many laboratories are not equipped to produce a specific absorbed serum, and yet the use of this or a bacterophage is essential for positive determinations.

Before the absorbed serum can be produced the laboratory must secure an authentic type culture of the organisms suspected of being present.

All this may take several months, and in the meantime diseased material from the original focus of infection may have been disseminated throughout

the country, so that when it is finally decided that the disease is new to the area, its limitation or eradication may no longer be possible.

To overcome this danger the speaker urged that consideration be given to the establishment at some central laboratory of a collection of specific absorbed sera for bacterial plant pathogens, and perhaps also for certain plant viruses.

Rapid identification would then be possible either by means of cultures forwarded to, or of serum forwarded from, the central laboratory.

Discussion: DR. WILTSHIRE explained that the Commonwealth Mycological Institute had an arrangement with DR. DOWSON at Cambridge for the identification of plant pathogenic bacteria, and that the possibility of preparing specific bacterial phages in dry culture for dispatch to plant pathologists overseas was being investigated.

DR. VASUDEVA and DR. CHAMBERLAIN said they would be interested in a scheme for rapid determination of plant bacteria and would use the facilities supplied.

MR. SAMUEL pointed out that in England a collection of bacterial plant pathogens was maintained on an official basis, as part of a culture collection for all groups of organisms. He thought it might be better to send the bacteria to this centre for indentification.

DR. CHAMBERLAIN wanted to be certain that there were such central facilities, and if there was provision for their continuity in the event of Dr. Dowson's retirement.

MR. SAMUEL assured him that this was so, and went on to say that there was a catalogue of culture collections in the British Empire issued by the British Commonwealth Scientific Office.

DR. BATES said that there was a Commonwealth Committee for culture collections that dealt not only with medical and industrial bacteria, but with plant pathogen material as well. He suggested that the matter be referred to this committee and the Resolutions Committee. This was agreed (See Resolution 7).

Meeting: Monday, 19th July, 2.30 p.m.

Chair: DR. W. F. HANNA

Subject: Eradication of established plant diseases

Paper: The eradication of sugar-cane diseases in Queensland

by C. G. HUGHES
(Bureau of Sugar Experiment Stations, Brisbane)
[Presented by C. R. Millikan, Australia]

The measures adopted for the control of cane disease in Queensland are in many respects unique, and for an understanding of them some knowledge

of the salient features of the industry is essential. Sugar-cane is grown only on the Queensland coast but through a wide range of conditions. There is one small mill between Brisbane and the Queensland - New South Wales border at a latitude of approximately 28° and from there the cane areas extend in separated districts to north of Townsville where it is practically continuous for more than 200 miles, culminating at Mossman in latitude 16°30'S., approximately 1,100 miles from Brisbane. Some 80 per cent. of the cane is produced within the tropics and all work is done by European labour. The unit of production is the farm, of which there are more than 8,000, although there are a few larger holdings in South Queensland worked as plantations. Regulations prevent growers supplying cane to any but a specified mill, and cane may be grown only on fixed areas on any one farm.

The control of diseases in Queensland (which used to enjoy a notorious reputation as the most disease-ridden cane country in the world) was rendered difficult by the large number of farmers in the industry, the miscellanea of canes under cultivation, and the wide range of soil and climate. On the credit side were the general high level of intelligence of the farmers, the awareness of disease arising from the severe losses which diseases had caused from time to time, and the willingness of the State Government to introduce enabling legislation. Control measures brought into operation by this Bureau may be grouped into two classes viz: legislative and direct. Both of course, take for granted a background of active research and experimentation in the individual diseases and the provision of a steady supply of the necessary disease-resistant varieties.

Legislative Control Measures.

The regulations in Queensland for the control of cane diseases are more comprehensive than in any other sugar-producing country, but as they are carefully framed and easily amended from time to time to suit changing conditions their enforcement has not presented undue difficulty.

The proclamation of quarantine against the free movement of cane planting material is one of the most important of the legislative measures aimed at reducing disease. The importation of varieties from abroad or from another part of Australia is the sole prerogative of this Bureau, which maintains a quarantine house in Brisbane well isolated from cane areas. The Bureau also controls the movement of cane from one part of Queensland to another, and for this purpose ten intra-State quarantine districts, generally with boundaries at well marked breaks in the sugar belt, have been established. There is also provision for placing in quarantine parts of a district, even down to single farms, and several small areas may occur within one major quarantine district.

These measures control the movement of plants from any diseased or suspect source, and are also used to define what varieties shall not be grown in diseased areas and to limit the crop cycle of susceptible varieties already growing. In addition, provision is made for the control of alternate hosts of cane diseases. This was particularly important in connection with downy mildew (Sclerospora sacchari) when plantings of the extremely susceptible maize had to be curtailed in the interests of the more important cane crop.

In common with most primary producers Queensland farmers are inveterate collectors of varieties, and if given free rein would grow hundreds of varieties in one district. The vast majority of these would be in very small amounts and would contribute little to the production of the district. Such a

miscellany of cane in commercial plantings is a serious danger from the disease point of view, and the number of canes has had to be limited on that account. This has been done by the introduction of a system of variety approval.

On January 1st each year a list of approved varieties is issued for each mill area. This sets out the varieties which may be grown by the farmers and must be accepted by the millers; all varieties not named are automatically prohibited. Some five to fifteen varieties are approved for each mill for planting in 1954 but, such is the range of conditions encountered in this State, the total number of varieties approved at one or other of the mills is thirty five (35). The list is made out by the Director of the Bureau of Sugar Experiment Stations who has to consider the behaviour of each variety with regard to diseases and insects and its agricultural and milling characteristics. Varieties are removed from the list when they become diseased or have lost popularity. Fairly heavy penalties are provided for planting, growing, milling, or even possessing any unapproved variety.

The actual implementation of control measures against diseases in the various districts is in the hands of Cane Pest and Disease Control Boards, which are administered by the Bureau. They are constituted wherever the disease or pest position warrants, and the territory of a single Board may be confined to one mill area or may embrace several contiguous areas. There are 21 such Boards in operation at present, each consisting of three representatives of growers and two of millers. Finance is provided by a levy on both grower and miller.

Direct Control Measures

Provision of disease-resistant varieties, selection of planting material, destruction of diseased plants, sterilization of knives, etc., and treatment of setts are the most important direct measures used in the control of cane diseases in Queensland.

The system of approved varieties could not be operated without a constant supply of new ones. In Queensland this is provided by raising seedlings locally and by the importation of commercial canes from abroad. The importance of each source varies from time to time, but in recent years the local seedlings have been the most important group, and for the 1953 crop provided more than 75 per cent. of the tonnage harvested.

The selection of clean sources of planting material with provision for forcing farmers to use only such cane is one of the most important factors in local cane disease control. Restriction on the right of a planter to choose his own plants is unheard of in most cane producing countries, but in Queensland its importance is recognised and very rarely has any prosecution to be launched.

Destruction of diseased plants may be on a small scale when individual diseased stools are "rogued", or on a larger scale by the enforced early harvesting or ploughing out of diseased fields. The timing of destruction programmes is very important and has to be based on a full knowledge of the disease and its transmission.

The sterilization of knives and the blades of the cutter planters is an important feature of control campaigns against such diseases as gumming and ratoon stunting, which are easily transmitted by mechanical means. The

handiest method of sterilizing is by heat either from a trash fire or from boiling water.

The treatment of setts is now well established in Queensland and may consist either of a treatment in hot water against such diseases as ratoon stunting and chlorotic streak or of the application of fungicides to the surface of the setts for the control of sett-rotting soil micro-organisms.

Control already attained with Various Diseases

What used to be major diseases in Queensland, viz., gumming and downy mildew, have now been eradicated, another (Fiji) appears to be approaching extinction and for two others effective control measures have been worked out and are in process of application. Each is discussed below.

Gumming disease (Xanthomonas vasculorum (Cobb) Dows.). There are definite records of gumming disease in Queensland as far back as 1892 and serious losses from it continued until the introduction of resistant varieties and the institution of rigid control of planting material. In South Queensland the standard varieties D. 1135 and M. 1900 S were heavily infected in the early 1930's but the substitution of the outstanding P. O. J. 2878 and Co. 290 led to the elimination of the disease, and at present M. 1900 S. is being grown once more. The substitution of resistant varieties in several northern mill areas also controlled the disease there. A recent serious outbreak occurred at Mossman in 1945 in S. J. 4 and H. Q. 426, which produced over 80 per cent. of the crop. A quarantine prohibiting the planting of these canes in the diseased and neighbouring localities was proclaimed and diseased fields were ploughed out after harvest. Gumming-resistant varieties such as Comus, Trojan, and Q. 44 were substituted and the last known diseased field was ploughed out in 1949. Continued surveys have not revealed any other focus of infection and already susceptible canes are being re-introduced.

Downy mildew (Sclerospora sacchari Miy.). Although downy mildew has been a serious threat to susceptible varieties at some time or another in most parts of Queensland it has never been recorded south of Bundaberg (25°S.). In that district, however, it became a major problem in the gumming-resistant P. O. J. 2878, and only constant attention prevented the loss of that valuable variety at a time when there was no adequate replacement cane in prospect. Control was generally obtained in the far north by the elimination of a few not very important canes and restricting planting material. In the Mackay district in central Queensland the disease prevented the early establishment of P. O. J. 2878, and plantings of that variety had to be rigidly controlled until the policy of roguing or ploughing out infected blocks eliminated the disease. The last report of downy mildew at Mackay was at the end of 1943 and constant inspections have not revealed any since.

At Bundaberg the control of downy mildew was a serious problem for the Board in view of the popularity of P. O. J. 2878 and the widespread desire to stand over that particular cane. However, the banning of the alternate host plant, maize, from the infected localities, the compulsory destruction of badly diseased crops and the consistent roguing of others (over 13,000 stools in one year), the rigid control of sources of planting material and the ploughing out of all contaminated fields within four years of planting led to a progressive reduction in the disease, and what is hoped to be the last diseased stool was rogued in 1950.

In the previous year an unexplained outbreak occurred in the Hambledon

mill area near Cairns, N.Q., which at the time had 70 per cent. of its cane lands under varieties susceptible to downy mildew. The discovery was made during routine inspections on January 27th, 1949. The danger was obvious since the wet monsoonal weather, expected to continue for the following three months, was ideal for the rapid spread of the disease through the susceptible varieties. One or two inspections per week were initiated immediately, but within four weeks daily inspections were commenced. The disease was found in two small blocks on adjoining farms separated by only a roadway, and in all 83 stools were rogued. At the same time all neighbouring cane was inspected and nearby farms placed under quarantine. The diseased fields were harvested and ploughed out as soon as possible, and the disease has not been seen in the district since.

Fiji Disease is caused by a virus transmitted by Perkinsiella saccharicida and has occurred only in mill areas in south Queensland. It was first recorded in Queensland in 1926 and was later found to be causing serious losses in fields in certain localities at Maryborough. Early measures taken against it were not very effective, as most of the varieties were susceptible to it. However, the decline in popularity of the susceptible P.O.J. 2878, the control of planting material, the destruction or roguing of diseased fields and the limitation of the cropping cycle have continued to the present time and it would appear the eradication of the disease is in sight. The intensive campaign has been in operation since 1938, when the Cane Pest and Disease Control Boards were constituted, and although at times more than 10,000 diseased stools were rogued at Moreton and over 7,000 at Bundaberg in the one year, the position has now so improved that only 62 stools altogether were rogued in the 1952-53 summer. The disease has been eliminated from two districts, viz: Maryborough and Childers, and there is every reason to hope that the remaining very lightly infected areas will soon be quite free.

Ratoon stunting and pineapple diseases. Ratoon stunting (probably caused by a virus) and pineapple disease (Ceratocystis paradoxa (Dade) Moreau) although quite dissimilar, are grouped together here because in each case the control measures have been worked out and it should be only a matter of time before each disease ceases to cause trouble. Ratoon stunting may possibly be eliminated but the fungus, although continuing to be present in the soil, should never cause any damage when the setts are suitably treated. Details of ratoon stunting are discussed in another paper before this Conference but mention should be made here of the activities of the Boards in all districts in supplying disease-free plants to their farmers. Hot-water units have been installed by them at the mills and within three seasons at the most, plants free from ratoon stunting disease should be available in sufficient quantities in all mill areas. The volume and weight of setts involved in any large-scale treatment present a mechanical problem of some magnitude, especially since the temperature of 50°C. must be accurately maintained for two hours if a good germination of disease-free material is to be obtained. Several Boards have treated more than 100 tons of plants and one has done nearly 500 tons to provide a nucleus of healthy planting material.

Pineapple disease is controlled quite effectively by the use of organo-mercurial compounds, one of the best of which appears to be ethoxy ethyl-mercuric chloride, although in Hawaii phenyl mercuric acetate has given satisfactory results. Treatment consists either of immersion of the setts in a solution containing 0.015 per cent. mercury immediately prior to planting, or spraying the individual setts with a similar solution as they drop from the knife of the cutter planter. The protection afforded is complete providing the

treatment is properly done, and the vexatious and expensive replantings or supplyings of "misses" in a gappy stand when plantings are made under adverse conditions, should no longer be necessary.

Paper: Attempts to eradicate other plant diseases from Australia

by C. J. MAGEE
(Department of Agriculture, New South Wales)

A number of diseases besides gumming (Xanthomonas vasculorum), downy mildew (Sclerospora sacchari) and Fiji disease (virus) have been successfully dealt with by eradicative methods in Australia. Details of some have been published, but uncertainty as to whether eradication of others is complete has withheld publication.

In addition to the cases I shall refer to mention should be made of the degree of success achieved in New South Wales during the war years in reducing to a low level certain diseases of vegetable crops namely, bacterial canker of tomato (Corynebacterium michiganense), halo blight of beans (Pseudomonas phaseolicola), and black rot of crucifers (Xanthomonas campestris). By co-operative work between the Department of Agriculture, seed merchants, and seed growers, involving seed treatment (particularly of stock or mother seed); crop inspection and in the case of beans, seed certification and elimination of infected lines, each of these diseases was reduced from major to minor status. I wish to record our experience that these diseases are easily amenable to control by concerted action. It is my opinion that if there was special reason and all concerned were prepared to tolerate restrictions for a few years these diseases could be eliminated from a country.

Eradication of Citrus Canker from Northern Territory of Australia

The eradication of citrus canker (Xanthomonas citri) appears to be the first successful attempt to deal with a plant disease by this means in Australia.

It first attracted attention in 1912 at Stapleton, 70 miles south of Darwin, but by 1916 it was known to be distributed in numerous small orchards of an acre or so and an assortment of citrus trees in town and homestead gardens. Many of the trees had been propagated at a single centre, the Botanic Gardens, Darwin, and it is thought that this was the focus of infection.

When the nature of the disease was realized all citrus trees and stocks in the Botanic Gardens were destroyed by burning as well as the infected trees at Stapleton. Proclamations were issued in 1915 and 1916 by the Commonwealth Government prohibiting the importation into Australia of citrus trees and fruits from all countries in which citrus canker was known to occur.

In 1918 an officer was appointed by the Commonwealth Government, with appropriate powers, to survey and arrange for the destruction of all infected trees in the Territory. Areas in which the disease was found were placed under quarantine restrictions (replanting) for a period of five years.

The efforts made in 1918 to eradicate the disease appear to have been only partially successful as a Commonwealth officer was sent to the Northern

Territory again in 1952 to make another extensive survey. New outbreaks of the disease were found and an Ordinance was issued requiring destruction of all citrus trees north of the nineteenth parallel. Growing of citrus trees on the Northern Territory of Australia was permitted again in 1925. Plantings were rigidly inspected for some years but no sign of the disease was found. An extensive survey of citrus in this region was made by a quarantine officer of the Commonwealth Department of Health in 1950 and it is now accepted that citrus canker has been eradicated from Australia.

Eradication of Boil Smut of Maize from New South Wales

Boil smut of maize (Ustilago maydis) was recorded for the first time in Australia in 1911, at a Government experiment farm at Bathurst, New South Wales. The disease recurred on the same site in 1919. In both instances steps were taken to effect eradication of the disease and no further maize was grown on the farm until 1929. Between 1929 and 1934 small areas of maize were grown each season on some portion of the infested area but no evidence of boil smut was observed.

In 1935, however, a few infected stalks were seen and a survey revealed the presence of six localised areas of infection. The whole crop was then cut by hand and destroyed by burning on the site. Infected plants were collected separately, bagged, and burned, the soil at the infested sites being drenched with formalin. Being Government land, arrangements were made that no further maize be grown either on or in the vicinity of the area where the disease occurred.

Surveys made in the Bathurst district in 1936 and 1937 showed no additional outbreaks, but in 1938 the disease was observed on a privately owned farm at Georges Plains (near Bathurst). The following year a wider survey revealed the disease on six additional privately-owned properties. The maize crops were cut by hand and burned on the site and the properties were quarantined for the further growth of maize under provisions of the New South Wales Plant Diseases Act. There was evidence that some at least of the farmers on whose land the outbreaks occurred had obtained seed from the Government farm at Bathurst.

In 1940 another survey of farms in the Bathurst district was made and the presence of boil smut was recorded on an additional three properties. The crops were burned and the farms quarantined. An extensive survey was made again in 1941 but no trace of the disease was found. The War intervened and no further surveys were made until 1947; again no evidence of boil smut was found. A further survey was made in 1948, special attention being given to observance of restrictions on the planting of maize on quarantined farms. In all cases the restrictions were being observed and no further outbreaks were found in surrounding farms. A survey was made in the Autumn of 1954 with the same result.

It is considered that boil smut has been eradicated but the known survival of the disease for 8 and 16 years between the three outbreaks in 1911, 1919 and 1935 dictate that surveys must be continued. The Bathurst district is a marginal one for grain production and maize is primarily a fodder crop so the quarantines do not impose great hardship on the landowners as sorghum and other alternate fodder crops can be grown there.

Control of Bunchy Top Disease of Bananas by Eradication Methods in New South Wales

Bunchy top (virus) made its appearance in New South Wales during the early stages of development of the banana industry, in all probability being introduced in 1913 by the planting of infected suckers imported from Fiji. By 1922 there were approximately 5,500 acres of bananas in New South Wales and bunchy top was then regarded as a serious menace. Unsuccessful attempts were made to check the spread of the disease by demarcating "buffer" zones between affected and unaffected areas and restricting the movement of planting material, but by 1925 the area under bananas had declined to 1,500 acres and production to less than one-seventh of the 1922 figures; most plantations were deserted.

In 1924-25 an investigation of the disease demonstrated that it was caused by a virus transmitted by the banana aphid Pentalonia nigronervosa. This aphid was shown to be an efficient vector but its part in the downfall of the industry was equalled in importance by the widespread planting of infected suckers. The virus was found to have a restricted host range, which meant that the situation was not complicated by an alternate host problem. In 1927 quarantine areas were proclaimed and restrictions placed on the movement of planting material. Registration of plantations became compulsory, Government permits were required to plant bananas in quarantine areas and eradication of diseased plants became obligatory. Representative banana growers were elected to committees under the title of zonal committees to assist the Department of Agriculture in the application of the regulations and Government inspectors were appointed to supervise eradication and to issue permits for restocking plantations. Later the duties of zonal committees were absorbed by the Banana Growers' Federation.

The control campaign early showed signs of being successful. The area under bananas expanded rapidly and bunchy top was maintained at a low level until 1932 when a banana "boon" commenced. Heavy planting during this and the following year led to overproduction, low prices, and general neglect of plantations. For a period bunchy top appeared to get out of hand. The introduction in 1936, however, of a supplementary scheme of control under which the Banana Growers' Federation, by a levy on production, maintained gangs of trained men to search for and treat diseased plants met the situation and overcame the problem created by newcomers to the industry, who were unfamiliar with the early symptoms of the disease. Since 1937 the annual incidence of bunchy top has declined from 2.69 plants per acre to 0.09 in 1952-53, when the area planted to bananas in New South Wales was 26,232 acres. The number of infected plants destroyed in 1952-53 was 2,427 compared with 3,654, 3,831, 5,385 and 5,565, respectively, in the preceding four years.

A stage has been reached in eradication at which the law of diminishing returns in operating strongly, and it is considered that under existing conditions within the industry total eradication of the disease will not be long delayed. Almost all outbreaks of the disease are now minor ones and by far the greatest number of them occur in plantations in which the disease has occurred previously. A problem of latent infection appears to exist in these plantations which could only be dealt with by destruction of whole plantations or parts of them. Many thousands of acres are now free from the disease, particularly in the Coff's Harbour district where there are about 7,000 acres in which the disease has never been reported.

The control of bunchy top of bananas by eradicative methods has also been successfully maintained in the south-eastern portion of the neighbouring state of Queensland.

Eradication of Cherry leaf spot from Nurseries in New South Wales

Cherry leaf spot (Coccomyces hiemalis) was first detected in Australia in a nursery near Sydney, New South Wales, in the summer of 1949-50. Nursery and field surveys were conducted and the disease was found in severe form in four other nurseries in or near Sydney. Each of the nurseries involved had received in 1948 cherry stocks imported from Holland and it is considered that the disease was introduced by this means.

Cherry leaf spot was proclaimed a disease under the New South Wales Plant Disease Act in 1950 and eradication was carried out in the five nurseries. The trees and stocks were ploughed out and destroyed by burning. The land was ploughed deeply to bury as much leaf litter as possible.

In the following season a further outbreak occurred in another Sydney nursery to which no imported stocks had been introduced and which had been free of infection the previous autumn. The source of infection of this outbreak is unknown. It is considered unlikely to have originated from airborne ascospores, as field and laboratory evidence indicated that no apothecia developed in buried and partly buried leaves in the unusually wet and mild winter of 1950. The trees were destroyed by burning and during the summer of 1950-51 all nurserymen who had received stocks imported from Holland voluntarily destroyed all unworked stocks, most of which had grown poorly.

Surveys conducted during the autumn of 1951 and each year since have failed to reveal any further sign of the disease in New South Wales and there is hope that the disease has been eradicated. In all about 12,000 trees and stocks were destroyed.

Eradication of Apple Scab from Western Australia

The eradication of apple scab (Venturia inaequalis) from Western Australia is especially notable in that a disease world-wide and costly to control is involved, which had penetrated deeply into a territory. This provides valuable information for estimating the range of ascospore dispersal of the pathogen.

Apple scab was first detected in Western Australia in the early autumn of 1930 at Manjimup. Severely scabbed fruit was found on about a dozen Cleopatra trees and the foliage of a large number of other trees of various varieties was also affected in a 17-acre orchard. A survey showed that neighbouring orchards were healthy. A few weeks later the disease was also found on a small number of Granny Smith trees at Porongorups, about 100 miles distant.

The Department of Agriculture of Western Australia acted promptly in both cases. All infected fruit and leaves, including windfalls, were gathered and destroyed by boiling or burning and all trees in the orchards were sprayed with Bordeaux mixture. When harvesting of unaffected fruit was completed all trees in the orchard were again sprayed with Bordeaux mixture. Later the whole area was sown down to subterranean clover and oats to provide a cover crop so that any infected leaves would not be blown about. The following spring the cover crop was ploughed in and the areas beneath the

trees were dug under by hand a fortnight before spur burst. Bordeaux mixture was applied at spur burst and again at pinking and petal fall. Surveys later in the year showed no sign of the disease at Manjimup and a few infected apples and leaves only at Porongorups. No fresh outbreaks of scab were found in Western Australia despite annual surveys until the late summer of 1936. This occurred also at Manjimup about 5 miles from the 1930 outbreak. Some 40 to 50 affected trees of several varieties were found distributed over $1\frac{1}{2}$ acres. The infection appeared to have spread in two more or less fanshaped areas in opposite directions from a badly affected tree. A month later another outbreak was reported at Mt. Barker, 100 miles from Manjimup. A fifth outbreak was detected about eighteen months later at Manjimup on a property adjoining that in which the third outbreak occurred in 1936. Each outbreak was dealt with in a similar manner to the first.

During the 1939-40 season the disease recurred in one of the properties at Manjimup and was found to have spread to some extent to an adjoining orchard. Also in this year new outbreaks were discovered at Kalgan River (near Albany) and at Jardee in the Manjimup district. Close surveys later revealed the disease in 21 orchards in the Manjimup district and three in the Albany district, although most of the infections were of a slight nature. The eradicative procedures that had been used previously were followed in the case of each outbreak, but closer supervision was given to the actual operations by officers of the Western Australian Department of Agriculture. In some cases flame throwers were used in the spring to assist in destroying leaves which had been blown about. Furthermore, orchards were not released from quarantine, and the entailed orchard inspection, cover cropping, and ploughing under of leaves, spraying and delayed cultivation in the summer, until they were free from scab for two full growing seasons.

Reviewing the position in 1944 the Department of Agriculture expressed hope that the disease had been eradicated. A new challenge came, however, in 1947-48 season when the disease was detected in 21 orchards and two nurseries. Investigations revealed that during the winter of 1947 large importations of apple nursery stock had been made from other Australian States where apple scab is well established. Many of the young trees were later found to have had bud scale infections. Drastic measures were adopted in dealing with the outbreaks, which included the destruction of the whole planting of a variety in which the disease was detected and the cutting back to basal dormant buds of any adjacent plantings of other varieties. In nurseries all stocks showing the disease were destroyed and the remainder were cut back to ground level and sprayed with a suitable fungicide. In all 3,700 young trees and stocks were destroyed and 19,000 trees and stocks were cut back.

There have been no further outbreaks of apple scab in Western Australia during the past five seasons and it is reasonable to conclude that the disease has been eradicated. Regulations were promulgated in 1948 prohibiting importation of nursery stock, finally removing the source which provided infection centres for previous outbreaks. The eradication is a remarkable administrative achievement although the climate of Western Australia, which is subject to dry periods in the summer months, possibly assisted the project. The apple crop of Western Australia in 1952-53 totalled 1,650,634 bushels, so that the annual saving to all concerned by eradicating this serious disease must be considerable.

Discussion: MR. MARSH (Long Ashton) asked first if phenyl mercuric

chloride spray has any adverse effect, say, on the buds or shoots, and second if such spraying would prevent bud scale infection.

DR. MAGEE replied that Hutton reported no damage even to leaves in his first trial with phenyl mercuric chloride. As yet this could not be guaranteed but it was thought to be good under their conditions compared with other compounds, which were quite toxic. There was no evidence of bud scale infection in New South Wales, and there seemed to be only one method of carryover of the disease.

MR. SAMUEL (United Kingdom) said he was very impressed and full of admiration for the courage with which this problem had been tackled in Australia. In the case of apple scab he could not help thinking of all the garden trees and derelict orchards in Great Britain and would like to know the position in this respect in say, Western Australia.

DR. MAGEE admitted that there were individual home-owners with apple trees, but in Western Australia backyard trees had been inspected. The Australian authorities were prepared to spend any amount of money so long as they had faith in the remedies.

MR. SAMUEL said he understood that South Australia had brown rot. He wanted to know if eradication had been successful.

DR. MAGEE replied that he could not give any accurate information on this matter, but he thought it had not been eradicated.

DR. HANNA (Canada) asked if Dr. Magee could describe the conditions in Western Australia. He wondered whether they were ideal or marginal for the development of apple scab. He thought there was a similarity between this area and some parts of Canada.

DR. MAGEE replied that if nothing had been done the disease would have been causing heavy losses, but scab was not severe every year. Accounts had been published by Pitman and Cass Smith in the Western Australian Journal of Agriculture.

DR. WELSH (Canada) referred to the irrigated districts in British Columbia where apple scab only appeared once in ten years. This situation had persisted until the last three years when there had been scab each year. An explanation of this new situation was still being sought.

DR. MAGEE said that a similar situation had arisen in New South Wales In an irrigated area where little trouble had been experienced from apple scab it had been a permanent disease for the last five years.

He went on to discuss the sugar-cane industry in New South Wales and described how disease in sugar-cane had been combated. He then read the following statement on the control of sugar-cane diseases in New South Wales, by E.G. Baber.

Paper: The control of sugar-cane diseases in New South Wales

by E.G. BABER
(Canadian Sugar Refining Company, Sydney)

The sugar industry in New South Wales has suffered severe epidemics

from a number of diseases, which have now been brought under control and the loss from them is negligible.

Diseases and Control Methods

In 1890-1900 and again in 1920-1925 gumming disease (Xanthomonas vasculorum) was widespread and losses so severe that the industry was threatened with extinction. In the first period the outbreak was brought under control by the use of resistant but inferior varieties. With the onset of the second epidemic, however, a more determined effort was made to eliminate the disease, again utilising in the beginning such resistant varieties as were immediately available.

It was first necessary to evolve a satisfactory form of disease resistance trial, by means of which, a number of wild varieties of sugar-cane were found to be almost immune. Repeated back crossing of some of them to commercial canes resulted in the production of high yielding, resistant varieties. With the extension of these new varieties infection was quickly reduced and gumming disease has now apparently been eliminated from the commercial sugar-cane areas; the last infected crop was recorded in 1941.

Fiji disease, due to a virus transmitted by the sugar-cane leafhopper, Perkinsiella saccharicida, was first recorded in New South Wales about 1900. It caused severe losses and would have eliminated the industry but for the control measures applied. At first these consisted of replacing the susceptible varieties with such resistant ones as were available but such a course was only partially successful as the varieties themselves were only moderately resistant and inferior in yield.

In 1920 it was decided to adopt systematic control measures such as roguing and seed selection combined with the growing of resistant varieties. It was soon realised that higher yielding varieties with more resistance would have to be obtained if the disease was to be brought fully under control and the industry remain economically sound. In 1926, therefore, a programme of breeding resistant and high yielding varieties was commenced. As with gumming disease suitable disease-resistance trials were devised, resistant parent canes discovered, and an intensive breeding programme instituted which resulted in the production of suitable highly resistant and commercially successful varieties.

Due to control measures direct losses amounted only to some 2 per cent. of the crop even at the peak of the epidemic, but the indirect losses due to the substitution of resistant but otherwise inferior varieties were at least equal if not greater. Infection has now been reduced to something of the order of one diseased stool per 50 acres.

The third major disease to be brought under control was leaf scald, Xanthomonas albilineans. Although not so widespread as either gumming or Fiji disease it nevertheless caused severe losses in certain susceptible but otherwise profitable commercial varieties. Control by seed selection was not very successful with this disease because of long dormancy so that it was necessary to rely mainly on resistant varieties. A programme of breeding was commenced in 1926 and a number of suitable resistant varieties were obtained which enabled the disease to be controlled.

The last outbreak occurred in 1950 and consisted only of a few diseased

stools. They were destroyed and, as all the varieties now being grown are resistant to leaf scald, there appears good prospects of the disease being eradicated.

Two other major diseases, which in the past have also caused severe losses, have now been brought under control, but, while losses from them have been eliminated, it does not appear possible to eradicate the diseases themselves.

Mosaic disease was widespread even before 1900 and caused severe losses which, although minimised by growing either resistant or susceptible but tolerant varieties, were still considerable. Systematic control measures (roguing diseased stools, seed selection, and the growing of resistant varieties only) were commenced in 1920 and by 1930, losses had been reduced to small proportions. The disease has now been almost eliminated from commercial cane crops. Unfortunately a number of alternate hosts exist, amongst which are several perennial grasses normally growing in close proximity to cane fields. As the disease is perpetuated in these grasses any return to susceptible cane varieties will mean re-infection of the cane fields and a recurrence of heavy losses.

Pineapple disease, Ceratocystis paradoxa, which usually attacks cane setts of susceptible varieties and prevents or reduces germination, has been completely controlled by dipping in organo-mercurial fungicides. However, this fungus can live saprophytically in the soil for long periods and there seems little possibility of eliminating it. Control measures, such as the above, will always have to be carried out when growing susceptible varieties.

Resistance to disease has been combined with high yield so successfully that the entire crop not only now consists of locally bred varieties, but the yield has been raised from 2.99 tons of sugar per acre in the period 1916-1920 to 4.50 tons in the period 1949-1953.

Organisation

The Colonial Sugar Refining Co. Ltd., (which operates also in Queensland and Fiji) is the only milling company in New South Wales and they have been responsible for devising the disease-resistance trials, breeding and testing of all new varieties, and the carrying out of disease control measures generally. These measures, which have proved satisfactory, have been on a voluntary basis between the miller and the grower for the betterment of the industry as a whole. The milling company had the technical knowledge, experience, and the realisation that good regular, healthy crops and prosperous farmers were an advantage to its business. The company provided the necessary staff, and the farmers also realised where their interests lay and co-operated fully.

Discussion (contd.): DR. WILTSHIRE asked what sort of staff was used for these eradication schemes.

DR. MAGEE replied that for inspection for maize smut a plant pathologist and a "fruit inspector" were employed. The latter was chosen for his approach to people and his reliability. He usually knew about the disease situation. Similarly for the banana eradication, retired-soldier types were preferred as being more suitable than younger men. There was not much to know, only rules to follow.

DR. WILTSHIRE asked how many they employed.

DR. MAGEE said that in New South Wales about 12 plant pathologists, who could be moved about the country. There were also about 100 "fruit inspectors". In the banana area there were about nine of them, employed at the expense of the banana industry. The bunchy top searchers were employed for their personality and had had no agricultural training, although some of them had been banana growers.

Paper: The eradication of diseases from New Zealand

by E. E. CHAMBERLAIN
(D.S.I.R., New Zealand)

Fireblight (Erwinia amylovora) is a serious disease of apples and pears in North America and was first found in New Zealand at Auckland in 1919. It was decided that an attempt should be made to eradicate this disease before it became widely distributed, and regulations were accordingly gazetted. The steps taken were as follows: (1) destruction of infected trees; (2) destruction of all hawthorn hedges and trees in those orchard areas where fireblight had appeared; (3) placing of an embargo on the passage of susceptible nursery trees from infected areas to clean areas. The disease when discovered was apparently well established and in spite of the steps taken it appeared during the following season in a number of localities within 100 miles radius of Auckland. Much of this spread was undoubtedly brought about by the distribution of infected nursery trees before regulations were gazetted. In spite of this set back the eradication campaign continued.

Because of its rapid rate of spread and wide distribution when first discovered and because of the prevalence of highly susceptible alternate hosts such as hawthorn and cotoneaster, which bridged the gaps between orchard areas, it is now obvious that eradication of fireblight was doomed to fail. However, the steps taken probably did reduce rate of spread as it took 17 years for the disease to reach Central Otago, our southernmost orchard area. Also the removal of most hawthorn hedges in orchard areas has probably had a lasting effect in keeping down the incidence of fireblight. Outbreaks in an orchard area must be dealt with promptly to prevent serious losses, so special teams are organised to cut out and destroy all infected spurs, twigs and branches. Except for odd seasons such measures are effective and losses are slight. It is estimated that in a normal year the average crop loss from fireblight does not exceed $\frac{1}{2}$ per cent.

Citrus canker (Xanthomonas citri) first appeared in New Zealand at Kerikeri, an isolated district north of Auckland, in 1937. Later it was found in a large commercial nursery, which distributed many citrus trees to domestic as well as commercial growers. An eradication campaign was organised immediately. Regulations governing eradication have been subject to a number of changes as follows:

1937 If infection severe, all trees in orchard barepoled.
If infection slight, infected tree plus 8 guard trees barepoled.

1938 If infection scattered in orchard, all trees barepoled.
If trees became reinfected, all trees barepoled.

1938 (contd.) If re-infection occurred a second time, all trees destroyed. (Later modified so that infected tree destroyed, and others bare-poled).

1940 First infection - infected trees destroyed
Re-infection - trees destroyed.

1940 Citrus canker regulations -
Infected trees plus guard trees destroyed. If a number of infected trees, all trees destroyed.

1941 Successful court action by a Tauranga grower prevented Government from enforcing destruction of other than diseased trees.

1945 Amended regulations.
Destruction of infected tree and all those within 36 ft.

1952 Regulations amended to include:
(a) In orchard: destruction of diseased tree only.
(b) In nursery: destruction of diseased tree plus all citrus within 200 yards.
(c) If re-infection occurs within 12 months all trees destroyed.
(d) Compensation to growers: based on age and crop: nursery trees flat rate.
(e) Embargo on movement of trees to and from declared infected area. (Infected area 500 yards radius from infected tree - in force for 2 years).
(f) Replanting not permitted within period of declaration (2 years).

Results: No citrus canker has been found at Kerikeri since 1946 and except for a wild citronelle none since 1941. Success resulted from strict eradication methods, but close inspection was possible in a small district such as Kerikeri.

At Tauranga the last infection was in 1953. In other places last infection was recorded in 1952 at New Plymouth. Under the present regulations there is some doubt as to whether complete eradication will be possible for the following reasons:

(1) There has been a wide distribution of trees from a nursery which has had 3 outbreaks of the disease - in 1940, 1946, and 1947.

(2) Inspection is attempted with the normal staff and under these conditions considerable difficulty is experienced in examining all citrus trees in New Zealand.

(3) It is doubtful whether the eradication regulations are sufficiently drastic.

Onion yellow dwarf was discovered in the Marshland onion growing area in 1938 and in the following season was found to be present in the seed-producing area of Blenheim. When discovered at Marshland the disease was already well established, being present in approximately 25 per cent. of the properties in the district. Investigations showed that crops were becoming infected mainly from onion seed crops, which carried the disease over from one season to the next. A certain amount of spread was also taking place from volunteer plants arising from discarded bulbs left lying on the ground.

In an attempt to control this disease the following regulations were gazetted:

(1) No onion seed crops to be grown in the district.

(2) After harvest, all stray, small, injured or otherwise worthless bulbs to be destroyed.

(3) Shallots to be lifted and sold for pickling only, and no further planting made.

(4) No jonquils to be grown in the vicinity of onion crops.

These regulations came into force in 1939 and in the following season the amount of infection was greatly reduced. The disease has not been observed at Marshland since 1940. In 1946 no trace of the disease could be found. It would appear therefore that onion yellow dwarf has been eliminated from the Marshland area. The success of control measures was largely due to the growers themselves who, realising the seriousness of the disease did their own policing. The disease has not occurred in any other onion growing area although it has appeared in isolated localities at intervals; in a domestic garden at Nelson in 1941, in an isolated commercial crop at Hastings in 1947, and recently it has reappeared in Blenheim.

Elimination of loose and covered smuts (Ustilago hordei and U. tritici) from malting barley. Loose and covered smuts used to be responsible for a considerable reduction in quality of our malting barley and hot-water treatment was introduced in an attempt to control these diseases. Treatment consists of a five hour pre-soak of grain at 60°F. followed by a five minute dip in water held at 127°F.

Trials on a commercial scale were commenced in the 1925-26 season, treated seed being supplied to farmers growing barley under contract to the Canterbury Seed Co., which practically controlled production of malting barley. The result of these trials was a 17 bushel per acre increase in yield, a marked improvement in grain, and absolute freedom from smut in a year when smut was particularly prevalent in untreated lines. These results were so promising that the Canterbury Seed Co. installed a hot-water treatment plant and treated sufficient seed to grow 560 acres in the 1926-27 season. Crops grown from treated seed were again free from smuts, yields were increased by over 26 per cent., and 15 per cent. more of the grain was 1st grade. About this time the system of treating nucleus lines in conjunction with use of seed one year removed from hot water treatment was introduced. In 1927-28, 1,041 acres were sown with clean seed and in the following year 4,331 acres. From this time on ample clean seed was available for growing and the malting barley crop has remained free from infection with smuts. The system has since been changed and at the present time hot-water treatment is carried out by the Crop Research Division, who hand the treated seed over to the Department of Agriculture, which has it grown under contract to provide nucleus lines. These nucleus lines are supplied to farmers who grow them under a certification system.

In this way both loose and covered smut have been eliminated from our malting barley crops.

It is still possible, although difficult, to find smut in odd barley crops not grown for malting.

Discussion: DR. HANNA (Canada) said that in Canada at Saskatchewan, by Provincial Law, an area was set aside where the farmers treated their seed by hot water and received a premium for this smut-free seed. They had found, however, that the first and second generation crops from treated seed gradually became reinfected. He wondered what Dr. Chamberlain's experience in New Zealand had been in this respect. Was continued treatment of seed necessary or did the progeny remain reasonably free. He added that in prescribed areas in Saskatchewan it was prohibited by law from growing barley with more than a trace of loose smut.

DR. CHAMBERLAIN said he was not sure how long the barley remained free, but smut did reappear after six or seven years if it was not treated. The system in New Zealand was to use the hot water treatment each year on the mother seed.

DR. CRUIKSHANK (New Zealand) said that the seed was treated every year, though some seed was never treated more than once. In some cases a small amount escaped treatment or in some lots the smut would not be completely eradicated and then there would be some transfer to the treated varieties. In their standard varieties there was no significant difference in the amount of smut in the four or five years during which the seed was sown prior to being issued for general cultivation.

DR. HANNA said that in Saskatchewan, after about two years, 40 per cent. of the seed was affected by smut.

DR. VASUDEVA (India) asked if they had tested the germination of the treated barley seed against that of untreated seed.

DR. CHAMBERLAIN replied that this had been done in 1924 or 1925 and there was no significant loss of germination.

DR. VASUDEVA asked if there was a less drastic treatment than the hot water. He said that in India the seed was put in cold water in the shade and dried in the hot sun in May and June. After this there was a definite fall in the germination of the barley but not of the wheat.

DR. CHAMBERLAIN said that in one trial with a pre-soak for five hours at 60^{O}F. followed by a five minute dip at 127^{O} followed by drying, the germination was not adversely affected.

DR. VASUDEVA said that it was the long dip in cold water that affected the germination.

DR. HANNA said that DR. RUSSELL at Saskatoon had made experiments on the effect of treatment on the germination of barley. He found a very definite reduction in the germination, some lots being affected more than others.

DR. BROADFOOT (Canada) said that in Canada they have set up an arbitrary figure in respect of distances to be maintained between treated seed and a field of ordinary barley. He asked if Dr. Chamberlain had any information about the distances required between fields of treated and untreated barley and wheat.

DR. CHAMBERLAIN said he had no information, but in a small country like New Zealand with small grain production practically all crops were

treated, so the distances of separation were less significant.

DR. CRUIKSHANK said that as far as certification was concerned no specific distances were laid down, and they were much more interested in varietal purity.

PROFESSOR MUSKETT (United Kingdom) said that some countries must be envious about the work being done in Australia and New Zealand. He could only give one instance of successful eradication from Northern Ireland — chrysanthemum midge, a pest, not a disease. In the United Kingdom all flax seed is treated with T. M. T. D. and apple scab is a tremendous nuisance, though its eradication has been considered.

DR. BATTS (Cambridge) referring again to loose smut said that the increase in yield and quality of the grain was so great after the hot-water treatment was introduced that he wondered if something other than loose smut was involved.

DR. CHAMBERLAIN said that he agreed with Dr. Batts that something else must have been involved, but it was an unknown factor.

DR. HANNA suggested that the hot-water method of treatment may embody some way of selecting high yielding types, and it should be investigated.

Paper: Eradication or attempted eradication of diseases in the West Indies

by R. E. D. BAKER
(Imperial College of Tropical Agriculture, Trinidad)
[Presented by D. Rhind, Colonial Dependencies]

Certain notable successes have been achieved in the eradication of well-established diseases, such as citrus canker in Florida, but in the West Indies not one of the five serious diseases which has appeared during the last thirty years has been eradicated. It may be profitable to examine why this is so.

The diseases to be considered are witches' broom (Marasmius perniciosus) of cacao; cacao virus; banana leaf spot (Mycosphaerella musicola); wither tip of lime (Gloeosporium limetticola); and leaf scald (Xanthomonas albilineans) of sugar-cane.

Witches' broom disease, indigenous to the South American mainland, was found in Trinidad in 1928, Tobago in 1939, and Grenada in 1948. In all three instances it was spread over a wide area before discovery. In spite of there being a vulnerable point, the green broom, in its life cycle and no alternate hosts, the rather half-hearted attempts at eradication were foredoomed to failure. In Grenada, by constant vigilance, the disease has been kept at a low level, but will obviously never be eradicated.

Cacao virus, discovered in Trinidad by Posnette in 1943 had again obviously been present for many years, but being mild, had been overlooked. Its symptoms, until seen by an expert on cacao viruses, had been confused with those of mineral deficiencies and with insect damage. When discovered it still only occupied a small part of the area under cacao and could very probably have been eradicated by felling, but this the planting community

would not agree to do, as cacao was valuable and the disease mild.

Banana leaf-spot was first recorded for the Caribbean area by Wardlaw in 1934 and by Stahel in Surinam about the same time. Within ten years it had reached almost every island and country from Surinam to Honduras. In this instance in Trinidad the discovery of the disease was made while it was probably still confined to one estate, but it was rapidly found that, short of destroying all bananas, sanitary methods were powerless to check its spread. It is now recognized that leaf-spot can only be controlled by spraying or by the use of resistant varieties.

Wither-tip of lime had followed a rather similar course some ten years previously, spreading rapidly throughout the island chain from Florida to Trinidad. No attempt at eradication was made, nor indeed would such an attempt have had any chance of success.

More recently leaf-scald of sugar-cane has appeared in British Guiana. Here again the disease had for some years been confused with other maladies of cane, and when discovered, again by a visiting scientist, was well-established from end to end of the cane belt. Eradication was clearly out of the question and control will turn on sanitary precautions in the field and the use of resistant varieties.

Two other diseases of sugar-cane should be mentioned; mosaic virus and gumming disease (Xanthomonas vasculorum). Both were formerly widespread in the West Indies, but the breeding of resistant varieties in Barbados has led to the virtual disappearance of gumming disease and of the southern Caribbean or less virulent strain of mosaic. A more virulent strain of mosaic in Jamaica is still unchecked. Only the plant breeder can take credit for these two examples of 'successful eradication'.

What can be learnt from this rather sorry tale? Firstly, that a surprising number of diseases has eluded the quarantine service and no doubt will continue to do so, as the service is powerless against wind-blown spores or casual smuggling of plant material.

Secondly, the Departments of Agriculture are not powerful enough either to detect the diseases quickly or to mount eradication campaigns. The junior officers are not well enough trained, and the well-trained officers too few in number; some colonies, indeed, being without a pathologist for long periods. Here the value of visiting officers, familiar with the diseases of other countries, is obvious.

Thirdly, given better facilities, is it likely that any of these diseases could have been eradicated, had they been found before they had become well established? In my opinion, those that spread rapidly by air-borne spores, like banana leaf-spot and wither-tip of lime, could never have been eradicated. Cacao witches' broom disease, although its spores are air-borne, might have been eradicated from a small or isolated area by very thorough removal of green brooms. Cacao virus disease could still be eradicated from Trinidad by a drastic cutting out campaign and leaf-scald of sugar-cane could probably have been eradicated from British Guiana if it had been found earlier.

What therefore is required for the future? Assuming that plant quarantine will not present a barrier to certain diseases, our second line of defence is to discover them quickly. Here it is important that agricultural officers

and even their most junior assistants and also the planting and farming communities shall be well enough trained to recognise new diseases. Their work must be supplemented by visiting officers, it being fatally easy to overlook something which you are not expecting to see, even when it is large and obvious. Then, lastly, having assumed the existence of an adequate reporting service, we need the trained pathologists with the knowledge and the powers to act quickly in emergency.

Discussion: DR. HOPKINS (C. M. I.) said that he would like to make clear the position regarding tomato spotted wilt, or 'kromnek' in Southern Rhodesia. At the last Conference he gave an account of the eradication of the virus by concentrating mainly on the destruction of infected dahlia tubers. He had heard since then that some eminent virologists had doubted the identity of the disease as he described. it. He wished now to inform the Conference that various samples of infected dahlias had been tested by Dr. McClean, of Pretoria, who had informed him that the virus was identical with that producing tomato spotted wilt in the Union of South Africa, and furthermore was lethal to the Bonanza variety of tobacco, which was widely grown in Rhodesia. What appeared to be final eradication had been accomplished by the destruction of all dahlia tubers both healthy and diseased in two infected nurseries in Bulawayo.

DR. BATES said that he supported Dr. Hopkins's statement about spotted wilt of tomatoes in Southern Rhodesia.

DR. RUDD JONES described the eradication measures against cypress canker [Monochaetia unicornis] in East Africa. By growing the resistant Cupressus lusitanica in place of C. macrocarpa and destroying all infected trees the incidence of disease fell 20 to 5 per cent. between 1949 and 1952. Since then no new infection of C. lusitanica has been seen.

Meeting: Tuesday, 20th July, 10. 0 a. m.

Chair: DR. E. E. CHAMBERLAIN

Subject: Fungicides

Paper: Antibiotics in plant disease control

by A. J. SKOLKO
(Division of Botany & Plant Pathology, Ottawa)
[Presented by J. W. Groves, Canada]

The urgent demands of medical practice for antibiotics of high purity, and incidentally correspondingly high cost, have monopolized both research and their production to the exclusion of any thought of potential agricultural uses until quite recently. A logical extension of the application of antibiotics in human and animal pathology to plant disease control has come with the realization that relatively crude preparations, cheaply produced and used in high dilutions, may provide more efficient control of certain plant diseases than chemicals currently in use, or even provide control where chemical treatments are ineffective. It is to be expected that the first step in this direction should be concerned with finding specific phytopathological applications for known antibiotics, especially against bacterial diseases. It may also be expected that new ones will be developed on the basis of their activity against plant pathogenic bacteria and fungi.

The use of antibiotics against plant diseases has been demonstrated in a

number of instances. The control of fireblight of apple (Erwinia amylovora) with thiolutin and streptomycin (Murneek, 1952) and of pear with streptomycin (Ark, 1953); crown gall of Bryophyllum (Bacterium tumefaciens) with penicillin (Brown & Boyle, 1945) and of pears and plums with several antibiotics (Hampton, 1948; de Ropp, 1949); apple scab (Venturia inaequalis) and early blight of tomato (Alternaria solani) with antimycin (Leben & Keitt, 1949); powdery mildew of bean (Erysiphe polygoni) with actidione (Felber & Hamner, 1948); cherry leaf spot (Coccomyces hiemalis) with actidione (Cation, 1953); halo blight of bean (Pseudomonas phaseolicola) with streptomycin (Zaumeyer et al., 1953; Mitchell, 1954); common blight of bean (Xanthomonas phaseoli) with streptomycin (Mitchell, 1954); soft rot of potato and carrot with streptomycin and (Pseudomonas fluorescens) with aureomycin (Bonde, 1953); and potato ring rot (Corynebacterium sepedonicum) with streptomycin (Van Schaak, 1948) is perhaps indicative of the broad possibilities of antibiotics in plant pathology.

Very few reports have been published on the use of antibiotics as seed treatment for the control of seed-borne diseases. Control of covered smut of oats with actidione (Henry et al., 1951) and of Victoria blight of oats with helixin B (Leben & Arny, 1952) indicate the effectiveness of antibiotics as seed disinfectants but, because of their instability in soil, antibiotics possess little protective value against soil-borne pathogens. Chemical seed dressings, such as the organic mercurials, are highly effective in eliminating inoculum on the surface of the seed or even superficial infection of the seed and at the same time provide protection against soil-borne pathogens. For the control of seed-borne diseases in which the pathogen establishes deep-seated infection in the seed none of the chemical seed dressings are effective. In the past we have depended on hot-water treatment for control of this type of infection but the special equipment and extreme care required are serious disadvantages. Goodman & Henry (1947) treated barley seed with subtilin for control of bacterial blight (Xanthomonas translucens). Ark (1947) used streptomycin for treating cucumber seed infected by Pseudomonas lachrymans (angular leaf spot). Sutton & Bell (1954) have found that black rot infection of swede seed (Xanthomonas campestris) could be controlled by soaking the seed for 30 minutes in an aequeous solution of aureomycin in dilution of 1:1000 and that the aureomycin on treated seed kept in storage for nine months retained its effectiveness against the pathogen. They also found that aureomycin treatment was compatible with arasan or spergon, which could be used for their protective value. A number of reports have appeared in which crude extracts, filtrates, or cultures have been used successfully to control seed-borne diseases but for practical purposes they need not be considered in detail here. The feasibility of controlling internal fungus infection of seed with antibiotics has been indicated by Wallen & Skolko (1950) by treating pea seed infected by Ascochyta pisi with antibiotic XG. In this connection it might be mentioned that the fungistatic effect of soaking infected pea seed in water alone was attributed to antagonistic substances produced by bacteria naturally associated with the seed (Wallen & Skolko, 1953). Such an explanation might account for the apparent control of loose smut of barley (Ustilago nuda) and wheat (U. tritici) by soaking the seed in water and in spergon solution (Tyner, 1953; Russell, 1953). If antibacterial and antifungal antibiotics can be found possessing low phytotoxicity, high disinfective properties at low concentrations, and stability in storage their use in plant disease control is assured.

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Paper: An antifungal substance produced by a strain of Bacillus subtilis.

by R. S. VASUDEVA
(Indian Agricultural Research Institute, New Delhi)

Fusarium udum causes a severe wilt disease of pigeon pea (Cajanus cajan) in India. Experiments carried out over several years consistently showed marked differences in the incidence of wilt in sterilized and unsterilized soils. Since the disease is soil-borne it was apparent that the normal soil microflora was responsible for the reduced disease incidence in unsterilized soils. Several fungi, bacteria, and actinomycetes were isolated from such unsterilized soils. Among these a strain of Bacillus subtilis showed marked inhibitory activity against the wilt fungus growing on potato dextrose agar. This inhibition was found to be due to the production of a diffusible substance by the bacterium. The substance exhibited maximum inhibition between pH 6.0 and 8.6.

Nutrition experiments showed that a dextrose-asparagin-phosphate medium was well suited for its production. Addition of minute amounts of manganese and iron to the nutrient liquid considerably enhanced the production of the substance. Optimum amounts were produced over a fairly wide range of temperature, i.e. 24° to 34°C. Earlier experiments showed that the principle was thermostable and, therefore, in all subsequent experiments unless otherwise specified the culture filtrate of the bacterium autoclaved at 10 lb. pressure for 10 minutes was used.

The growth of F. udum was not stopped in soil-maize, sand-maize, and sand-soil-maize columns impregnated with the culture filtrate, but it was retarded. Greater inhibition was noticed in the case of sand-maize than in either soil-maize or sand-soil-maize. Loss of activity of the inhibitory principle in soil was not due to either the soluble salts or organic matter present in it. Immediate loss of activity of the culture filtrate was observed when it was passed through beds of soils of varying pH values (5.0 to 9.2). In each case the pH of the filtrate, after passage through soil, approximated the pH of that soil; readjusting the pH to its original value did not restore the activity of the culture filtrate. These results indicated that the observed loss of activity might be due to the adsorption of the prinicple by the clay fraction. This was confirmed in an experiment in which sand mixed with different proportions of clay was used in place of soil.

In Petri dish tests the culture filtrate of B. subtilis was inhibitory not only to F. udum but also to certain other pathogenic fungi. The inhibition of Alternaria solani particularly attracted our attention since it is the causal organism of early blight of potato, a fairly widespread disease, and the

fungus itself is well-suited for assay purposes by the slide-germination technique. Further studies on the antibiotic were, therefore, carried out using A. solani. The untreated culture filtrate completely inhibited the germination of A. solani spores. This inhibitory activity was considerably reduced but not completely lost on autoclaving the filtrate at 10 lbs for 10 minutes. The autoclaved filtrate still showed good fungistatic activity and was fungicidal when A. solani spores were soaked in it for prolonged periods.

The autoclaved extracts also possessed to a marked degree the important physiological effect of inducing 'bulb' formation on spores and hyphae of A. solani, F. udum, Physalospora tucumanensis, Botrytis allii, Cunninghamella echinulata, and Lophotrichus ampulus. Bulb-formation in the unheated filtrate was almost negligible though not completely absent. It has not been possible so far to separate the inhibitory and bulb-forming activities. (For the sake of convenience the term 'bulbiformin' is used to designate the complex factor or factors involved.) The bulb-forming activity could be assayed by using A. solani as the test organism, the number of bulbs produced on the spores being proportional, within the limits of experimental error, to the log. concentration of bulbiformin. The amount of the substance present in 1 ml. of the test liquid which induces the production, on an average, of 10 bulbs per 100 spores of A. solani strain No. 175 (Indian Type Culture Collection), mounted in 1 drop of such liquid on a slide and incubated for 24 hours at 25°C. has been taken as the unit of activity.

Bulbiformin can be concentrated from the culture filtrate by adsorption on kaolin, elution with acid ethanol (pH 4.8), and subsequent removal of alcohol, or by solvent extraction with isopropanol. It is also soluble in water, methanol, n-butanol, and pyridine.

On the basis of the studies described here and others with detached potato leaf cultures, in which infection by A. solani was inhibited experiments were carried out with potted potato plants in which the filtrate was applied to the leaves simultaneously with or before and after the spore suspension was used. Infection was markedly controlled under certain conditions. Experiments with cut shoots dipped in the filtrate similarly reduced infection, indicating that diffusion through the plant was possible.

In tomato also infection of leaves by A. solani was prevented, not only when the culture filtrate was applied to the leaf, but also when it was fed to the roots in solution culture. The filtrate proved to be phytotoxic at higher concentrations, the plants wilting in two days. Transverse sections of the wilted plants at the collar region showed necrosis of the cells even though the fungus was absent. In sand cultures also incidence of infection was appreciably reduced. Guttation fluid collected from such plants maintained for brief periods in a humid atmosphere inhibited the germination of A. solani spores, while no such inhibition was noticed in the case of the controls, thus indicating active uptake and systemic translocation of the antibiotic within the plant. There was some indication that similar uptake could take place in sugar-cane, for setts dipped in the culture filtrate and subsequently inoculated with P. tucumanensis showed reduction in the spread of red rot within the setts.

The antibiotic principle also inhibited infection of host plants by the viruses of petunia, chilli, and periwinkle mosaic. Petunia and periwinkle inoculated with mosaic viruses immediately after treating with the antibiotic showed partial infection, while inoculation 24 hours after adding the antibiotic to the virus preparation gave no infection. This was interpreted

as meaning that inhibition was due to the inactivation of the viruses in vitro rather than to a change in the susceptibility to the host cells at the point of application.

Paper: Use of antibiotics in control of stone fruit blast (Pseudomonas syringae van Hall)

by E. E. CHAMBERLAIN
(D.S.I.R., New Zealand)

Promising results in control of stone fruit blast with streptomycin is of particular interest because it may provide means of controlling a disease against which the usual plant therapeutants are ineffective.

The investigations outlined here were carried out by D. W. Dye, a bacteriologist and M. H. Dye, a microbiologist specializing in control of plant diseases by antibiotics, both of the Plant Diseases Division, New Zealand.

Stone fruit blast, which is widespread in New Zealand and which has been present in the country for many years, is a serious disease as it causes the death of young trees. Trees up to six years old are the most susceptible. In some areas, particularly Central Otago, considerable difficulty is being experienced in establishing new plantings. In some blocks of young trees over 90 per cent. have been killed in one year. Greatest losses occur amongst trees which are infected when they leave the nursery. All stone fruit species are susceptible and the causal organism Pseudomonas syringae, which closely resembles P. mors-prunorum, also infects lilac, poplar, willow, citrus, pear, rose, grape, and other plants.

The pathogen was identified in 1951 and experiments on control initiated in 1952. In preliminary trials 23 proprietary therapeutants and five antibiotic substances were used at varying concentrations. Both the proprietary therapeutants and antibiotic substances were applied as foliar sprays to young peach trees which had previously been injured at a number of points on the stems. As soon as the treated trees were dry they were sprayed with a broth culture of a pathogenic strain of P. syringae. After treatment all plants were held under conditions suitable for development of the disease.

A few of the proprietary therapeutants gave some degree of control, the most effective being S. R. 406 (captan) which at 2 lbs per 100 galls reduced infection from 90 per cent. in untreated checks to 53 per cent. At higher concentrations it caused plant damage and was less effective in controlling the disease. Much better results were obtained by antibiotics. Streptomycin at 250, 500, and 1000 μgm./ml. reduced infection to 16, 12, and 10 per cent., respectively, compared with 89 per cent. in checks. Aureomycin at 1000 μgm./ml. reduced infection from 97 per cent. to 20 per cent. but was not effective at lower concentrations. Not only did streptomycin produce a reduction in the number of lesions but it also reduced the size of those lesions which did develop. At 1000 μgm./ml. it reduced lesion length to 1.3 cm. compared with 3.1 cm. on check trees.

Following these initial experiments two further screening trials were carried out, (1) in vitro tests with seven antibiotic substances, and (2) in vivo tests using the same seven antibiotics and the seven proprietary therapeutants, which had given the most promising results in the preliminary

trials.

(1) In vitro tests. In these trials it was found that aureomycin was the most effective compound, growth of P. syringae being inhibited at concentrations between 0.1 and 0.6 μgm./ml. after 24 hours incubation. The order of effectiveness of the remaining six antibiotics was as follows, growth inhibiting concentrations at 24 hours incubation being given in brackets: streptomycin (0.3 to 0.8); dihydro-streptomycin (0.8 to 1.0); terramycin (0.6 to 2.0); polymycin (1.0 to 4.6); thiolutin (10.8 to 15.4); and chloromycetin (24.4 to 30.8). In addition to the experiments on the bacteriostatic action of antibiotics, in vitro studies on the bactericidal action of streptomycin were also made. The results obtained suggest that streptomycin is bactericidal for P. syringae.

(2) In vivo tests. The experimental method used was the same as that of the preliminary trials, there being 120 inoculation points in each test. None of the proprietary therapeutants tested gave any marked degree of control although S.R. 406, tetroc, and Bordeaux mixture all significantly reduced infection at one of the three concentrations employed.

All antibiotics except thiolutin significantly reduced infection when applied at 1000 μgm./ml. Streptomycin and dihydro-streptomycin were the most effective, both reducing infection from 91 per cent. to 2 per cent. when applied at 1000μgm./ml. Streptomycin significantly reduced infection at all concentrations from 50 μgm./ml. to 1000 μgm./ml. It was again shown that lesions which developed on trees sprayed with high concentrations of streptomycin and dihydro-streptomycin were not phytotoxic at any of the concentrations used. These results are considered to be very promising since the injuries were relatively severe, concentration of inoculum was high (between 2.5×10^9 and 5.7×10^9 bacteria per ml.) and conditions for infection were ideal.

FIELD TRIALS. In addition to the above screening trials experiments were carried out in the field at Auckland during the winter of 1953. In one trial trees were sprayed at mid-winter and in another at bud-movement. Young peach trees were injured at up to 40 points per tree and then sprayed with a heavy inoculum of P. syringae. Inoculated trees were sprayed with S.R. 406 (2 lbs. per 100 gals.), Bordeaux mixture (2-4-100),and streptomycin at 250 μgm./ml. The following results were obtained:

Winter treatment

Check: 70 per cent. infection
Bordeaux mixture: between 50 and 60 per cent. infection
S.R. 406: between 50 and 60 per cent. infection
Streptomycin: 15 per cent. infection

Bud-movement

Check: 83 per cent. infection
S.R. 406 and Bordeaux mixture: between 60 and 70 per cent. infection
Streptomycin: 26 per cent. infection

In check trees there was a rapid increase in size of the lesions and many trees were killed. On trees treated with streptomycin lesions remained small and only a few twigs and branches were killed.

A number of trials, using streptomycin under normal orchard conditions, have been laid down in Central Otago to determine (1) the number of spray applications required to control the disease, (2) the best times for application, and (3) the possibility of securing economic control. Concentrations of 100 μgm./ml. and 250 μgm./ml. are being used. Results are not yet to hand.

In New Zealand most of the young trees for commercial planting are produced at the nursery of the New Zealand Fruitgrowers' Federation. As greatest losses are experienced when young trees become infected before they leave the nursery trials are being carried out at the Federation Nursery to determine whether trees can be kept free from infection during propagation. Streptomycin at 100 μgm./ml. is being used.

STUDIES ON SYSTEMIC ASPECTS OF STREPTOMYCIN. Work on systemic aspects of streptomycin has been carried out on peach trees. No evidence has been secured to show that streptomycin applied to the foliage of plants is absorbed through leaves or stems. It is, however, readily taken up through the roots and becomes systemic in the tree. It is also readily taken up when injected into the tree by the method used by Roach for injecting trees with trace elements. Streptomycin introduced into a tree by injection does not become evenly distributed throughout the tree. When introduced either through the roots or by injection streptomycin after a time tends to become concentrated towards the growing points. At high concentrations it gives complete control of stone fruit blast, but is phytotoxic, causing cessation of growth. At low concentrations it is not phytotoxic, but gives only partial control. It is possible that there is a concentration at which it is not phytotoxic and yet gives complete control, but this concentration has not yet been ascertained. Streptomycin appears to be stable in the tree and has been shown to be present for periods of up to five months.

CONTROL OF BEAN HALO BLIGHT WITH STREPTOMYCIN. Although this discussion is primarily concerned with the use of antibiotics for control of stone fruit blast I would like to make mention of experiments by D.W. Dye on the control of bean halo blight (Pseudomonas phaseolicola) with streptomycin.

Young bean plants growing in the field were sprayed with a broth culture of P. phaseolicola. The disease was allowed to develop before control treatments with Bordeaux mixture, phygon, and streptomycin were commenced. Plants were sprayed three times at intervals of two weeks. At the time of the first spray 91 per cent. of the plants were infected. Results were as follows:

	Percentage pods infected	No. pods picked
Check:	21	2156
Phygon: 1 lb. per 100 gals.	22	2078
Streptomycin: 100 μgm./ml.	12	5421
Bordeaux mixture: 3-4-50	2	5611

The weight of pods corresponded very closely to the number of pods. The onset of dry, hot weather prevented good development of infection at the pod stage. At the first picking the percentage of infected pods on streptomycin treated plants was as high as in checks, but the degree of control improved at each successive picking, suggesting that streptomycin would have been very effective if applied before plants were exposed to infection. Bordeaux mixture gave consistently good control at all pickings.

Discussion: DR. WELSH (Canada) asked Dr. Chamberlain if he had found any adverse effect on fruit flavour, stating that in Wisconsin antibiotics applied with a spray gun had produced a bitter flavour in cherries.

DR. CHAMBERLAIN replied that in New Zealand they were mainly attempting to eradicate bacterial diseases from nursery stock so that guaranteed healthy trees only could be distributed, and were not at the moment concerned with protecting trees in fruit.

In answer to another question he stated that they had no information on whether P. syringae persisted through the summer on leaves, but in view of the findings at East Malling with P. mors-prunorum he intended to investigate the matter in New Zealand.

DR. MILLIKAN (Australia) said that in the United States fireblight of apples and pears was strikingly controlled by streptomycin, which, however, injured the leaves, but when mixed with indol-acetic acid no injury occurred.

DR. CHAMBERLAIN said that there was no foliar damage by streptomycin to peaches, and it was their experience that phytotoxicity of antibiotics was very specific.

In work on halo blight of beans he had found that streptomycin became systemic, but he had no evidence of this with peaches, which again emphasized the specific nature of the antibiotic.

DR. CROWDY asked whether the leaves from which streptomycin was recovered five months after treatment were on the tree at the time of treatment because it had been suggested that there might not be translocation in some plants.

DR. CHAMBERLAIN replied that the leaves were not on the tree at the time of spraying and that this again emphasized the uncertainty of antibiotics. For what might happen in one host was no indication of the reaction of another.

DR. CROWDY confirmed this by reference to translocation in cucumber, tomato, and broad bean.

DR. KEYWORTH asked for information on the weathering properties of antibiotics applied to leaves.

DR. CHAMBERLAIN replied that trials had only recently commenced in New Zealand where they were applying streptomycin as a therapeutant spray hoping that it would become systemic. They had no information on weathering.

Paper: Recent work on systemic fungicides and bactericides

by P.W. BRIAN
(Imperial Chemical Industries)

INTRODUCTION

"The earlier work on systemic treatment of plant disease failed to carry complete conviction, though, in the light of more recently acquired knowledge, it seems likely that many of the earlier claims were in fact well-founded. I

do not intent today to deal with this earlier work which has been adequately reviewed.[1,2] I propose to use a selection of more recently reported results to illustrate some methods of approach to the problem of systemic treatment and indicate some of the difficulties likely to be encountered. I am not qualified to suggest what their place in practical disease control is likely to be; perhaps on the basis of my remarks you may be better able to form your own conclusions on that aspect.

MECHANISMS OF FUNGICIDAL ACTIVITY

I can deal with the subject more easily if if first say a few words about fungicidal activity in general. The points I shall make equally well apply to antibacterial activity.

As you well know, research on fungicides has until recently been carried on at what may be called a superficial level. Investigations have been largely confined to an empirical search for new compounds toxic to fungi or to finding means of formulating known toxicants in such a way as to improve their performance. I would not wish for a moment to underestimate the benefits that have accrued from such investigations. The point that I want to make is that at this level of investigation, or even at the somewhat deeper level of systematic examination of substances structurally related to known toxicants, we are accepting the criterion of toxicity to fungi without any great attempt to understand the mechanism of fungicidal action.

More recently, greater emphasis has been placed on studies of how fungicides work, on a biochemical level, in terms of enzyme chemistry. At this level, fungicides appear as substances which interfere with the normal metabolism of the fungus. Most metabolic processes consist of a series of enzyme or enzymes. A fungicide may therefore cause metabolic disfunction by inactivating or inhibiting some key enzyme, by simulating some metabolic and so blocking enzyme surfaces with inappropriate molecules or by denying some important metabolite to the organism by chemical or physical means.

Perhaps a few examples may make this clearer. Mercurial fungicides, whether inorganic mercury salts or organo-mercurial compounds, are believed to owe their activity to their great affinity for sulphydryl groups; many enzymes (e. g., succinic dehydrogenase, urease, triose phosphate dehydrgenase) bear active -SH groups and if these are bound by a mercurial the enzyme is inactivated. The activity of unsaturated ketones and lactones has been similarly explained. Such compounds are themselves competitively inactivated by appropriate sulphydryl compounds, such as cysteine or glutathione.

Dinitro-o-cresol, long used by biochemists as a respiratory enzyme inhibitor, is believed to act in that capacity by inhibiting the turnover of nucleoprotein phosphorus. Its antifungal activity is probably similarly explained.

Some sulphonamides are actively antifungal as well as antibacterial. The mechanism of their antibacterial activity has been investigated by Dr. D. D. Woods of Oxford, and this work has opened a new approach to problems of toxic action. In the metabolism of many bacteria a substance known as folic acid plays an essential part. One of the substances from which the biosynthesis of folic acid starts is p-amino benzoic acid (PAB). Some

bacteria can synthesise this PAB themselves, others need to be supplied with it. The sulphonamides are structurally related to PAB and by taking its place on an enzyme surface prevent the biosynthesis of folic acid. This view is supported by the fact that sulphonamide drugs are antagonised competitively by small quantities of folic acid. The antifungal activity of sulphonamides is probably based on a similar mode of action[3].

The antifungal activity of 8-hydroxyquinoline is probably associated with its affinity for heavy metals, which may be thus denied to metal-activated enzymes in the fungal cell.

PENETRATION OF FUNGICIDES TO AN APPROPRIATE "SITE OF ACTION"

In addition to having some specific inhibitory effect, an effective fungicide must be able to penetrate to its appropriate "site of action". As a rule, it must be able to penetrate the osmotic barrier of the fungal cell. There is much evidence, for instance, that neutral molecules penetrate more readily than charged ions. Weak acids and bases penetrate more readily in the undissociated form; for instance, it has long been known that such phenols as dinitro-o-cresol are more active in spore germination tests at low pH than at higher pH, and that the activity at any given pH is related to the degree of dissociation. It is not necessarily true, however, that the form in which a molecule penetrates most readily is necessarily the form in which it acts once it has penetrated the osmotic barrier. My colleague J. F. Grove[4] has shown, in the case of the antibiotic gladiolic acid, which can exist in several tautomeric forms, that in all probability the most rapidly penetrating molecular species is the undissociated lactol form, whereas the active inhibitory species in the fungal cell is the open-chain anion. I mention this point here because where such a fungicide is to be considered for use systemically, one must consider that it may be that the form which most readily enters the plant, the form which is most readily translocated and the form which most readily penetrates into the cells of a fungal pathogen, may not necessarily be identical.

PROPERTIES REQUIRED OF AN EFFECTIVE SYSTEMIC FUNGICIDE OR BACTERICIDE

Application of a fungicide or bactericide systemically introduces several additional problems. I propose to mention a few of these at this point before dealing with experimental work on systemic treatment.

TRANSLOCATION. An effective systemic fungicide must be taken up at some suitable plant surface (viz. leaves or roots) and readily translocated. It might be thought that high water-solubility was an important property to achieve uptake and translocation. This is not necessarily so; griseofulvin, an antibiotic which we have found to be an effective systemic fungicide, is only soluble at 25° to about 30 μg./ml.

PHYTOTOXICITY. The more intimate contact between host tissue and fungicide, achieved by systemic application, greatly increases the likelihood of damage to host tissues. There are weighty reasons why a fungicide used in this way may prove to be phytotoxic. As I have already said, a fungicide usually acts by interference with some metabolic process, catalysed by an appropriate enzyme system, in the fungal cell. The basic metabolic processes in cells of every kind of organism tend to be similar, and the more closely related two organisms are, the more similar they are in biochemical constitution. While an insect and a plant may have some metabolic processes

in common, there will be an appreciable number that are different, so it is not excessively difficult to find an insecticide which is harmless to plants and so can be used systemically. The metabolic differences between a higher plant and a fungus are much smaller and it will be correspondingly more difficult to find a substance which will inhibit a metabolic process essential to the fungus without similarly affecting the host plant. Looking at the problem from this point of view, an effective systemic fungicide must interfere with some specifically fungal metabolic process and have no comparable effect on plant metabolism. Unfortunately our knowledge of fungal metabolism is not sufficiently detailed at present to give us many leads to investigation.

Achievement of specificity by this approach is perhaps a counsel of perfection, and effective substances may be found without going to such lengths. Let us consider for a moment the fungicides and their modes of action which I mentioned at the beginning of this paper. Immobilisation of -SH groups by organo-mercurials, and interference with nucleo-protein metabolism by DNOC would be expected to induce toxicity to all types of cell, and these two kinds of compound are, in fact, highly phytotoxic as well as antifungal, and it is not surprising that neither have been proposed for use systemically. Sulphonamides also are toxic not only to fungi and bacteria, but to plants as well.[3] Nevertheless, sulphonamides show promise as sytemic fungicides.[5,9] Similar considerations apply to 8-hydroxyquinoline. It appears probable, therefore, that the cell-sites sensitive to a given toxicant may, in some sense, be more exposed in the fungal cell than in the host plant. It is possible that in many cases it will be possible to use as systemic fungicides substances intrinsically toxic both to host and parasite, where the margin afforded by such differential accessibilities of sites of action is sufficient. Nevertheless for absolute safety substances affecting some specifically fungal metabolic process seem most promising.

METABOLIC ALTERATION OF THE FUNGICIDE IN THE PLANT. A fungicide applied to a fungus may be chemically modified as a result of metabolic activity of the fungus. For instance, the dry Bordeaux deposit is solubilized by complex-formation with organic acids secreted by the fungus, and copper penetrates the fungal osmotic-barrier in that modified form.[6] The marked tolerance of certain antifungal phenols and quinones exhibited by Macrosporium sarcinaeforme has been attributed[7] to oxidation of the phenol or quinone by a phenoloxidase (laccase) secreted by the fungus. If such changes can be induced by the small cell-volume present in a spore suspension, it would seem likely that in the presence of the relatively bulky tissues of a plant, a systemic fungicide might be modified as a result of metabolic activity of the plant cells. Indeed, it is known that some toxic substances (e.g., ethylene chlorhydrin, chloral hydrate, o-chlorphenol) are changed to less toxic substances, usually glycosides, in plant tissues.[8] We must consider the possibility, therefore, that a systemic fungicide might be changed to some non-fungicidal substance in plant tissues. It is also possible that the plant might so alter a substance as to make it less phytotoxic but still antifungal, or might even enhance the antifungal activity. We must bear in mind, too, the possibility that a foreign substance introduced into a plant might actually decrease resistance to disease. For example, ethyl alcohol has been shown to reduce the resistance of tomato plants to Fusarium wilt.

These various complicating factors illustrate the greater complexity of the system we must expect to deal with when fungicides are applied systemically. I should like now to make these points a little more concrete by considering the behaviour of some substances which have been found to possess systemic fungicidal activity.

SUBSTANCES RELATED TO PLANT GROWTH REGULATORS

Crowdy and Wain[10] based their approach to the discovery of systemic fungicides on two premises:- (a) that certain plant growth regulators were known to be translocated freely in plants; (b) that some of the plant growth regulators were known to be antifungal, though not markedly so. They examined a series of aryloxyalkyl carboxylic acids to see whether it was possible, by modification of the molecules, to reduce phytotoxic activity while at the same time enhancing antifungal activity. They found that chocolate spot of broad beans, caused by Botrytis fabae, could be significantly reduced in intensity by the application of such substances to the roots or leaves and that systemic activity was roughly paralleled by in vitro fungistatic activity. They considered it reasonable to assume, though it was not directly proved, that these substances were being translocated and acting as systemic fungicides unchanged. They were able to find compounds which were comparatively non-phytotoxic. The degree of control of chocolate spot achieved was highly significant, but by no means complete. Subsequently, Byrde, Crowdy, and Woodcock[11], in a study of some chlorine substituted naphthols and naphthyloxy-aliphatic carboxylic acids, found active compounds, but in general the substances most antifungally active in vitro were also most phytotoxic. When systemic activity was studied some interesting anomalies were discovered. The best disease control in vivo was given by certain 3:4-dichloro-2-naphthyloxy compounds which were among the least antifungal in vitro. Several explanations for such a result could be offered, but in the absence of further collateral evidence it is perhaps unwise to speculate. Davis and Dimond, [12] who have tested a number of compounds of this type for control of Fusarium wilt of tomatoes, also found that systemic activity was not necessarily correlated with in vitro antifungal activity, and preferred to explain systemic disease control as a result of altered host metabolism by the compounds used. Be that as it may, the work with plant growth regulators has fully established the validity of Crowdy and Wain's approach, viz., selection of compounds of a type known to be translocated in the plant. At the same time it appears likely that the compounds so far uncovered are too liable to be phytotoxic to be considered for practical use.

METABOLITE ANALOGUES

I mentioned earlier that some bactericides and fungicides are believed to owe their activity to structural similarity to essential metabolites, and I instanced the case of the sulphonamides. The sulphonamides are also phytotoxic, the mechanism of action being similar to that of their antifungal activity. Nevertheless sulphonamides have been successfully used by Hotson[5] for systemic control of wheat stem rust (Puccinia graminis tritici). Sulphadiazine was the most effective compound, and did not cause serious damage to the host at dosages which gave effective disease control. Control was achieved by application to soil and by application to foliage. Disease control was ineffective if treatment was followed by treatment with p-aminobenzoic acid.

As I have already suggested, this result perhaps indicates that the folic acid synthesizing system is somehow more accessible in the rust fungus than in the wheat plant. Alternatively it may be that the rust depends on the host for synthesis of PAB or folic acid, and if biosynthesis is restricted in the host by the presence of a sulphonamide, the host's requirements are satisfied before those of the parasite. Whichever is the case, the result is that fungicidally effective but non-toxic dosages can be found.

It is worth noting that the ready translocation of sulphonamides may quite possibly be associated with the fact that they are structurally related to a normal metabolite of the host - PAB. Indeed, the case of the plant growth regulators is another example of this, the plant growth regulators being structurally related to indoleacetic acid, a natural growth regulating hormone in plants.

ANTIBIOTICS

Those substances which we call antibiotics include substances of many kinds of chemical structure and of varied biological activity. The majority of antibiotics are generally toxic; certainly many are quite markedly phytotoxic.[13,14] They are attractive substances to work with, as they can often be bioassayed with some degree of specificity, and the process of translocation can in such cases be conveniently followed.

The use of antibiotics in plant disease control has recently been reviewed,[15] so I shall only deal with selected aspects here.

Considering for the moment only well-characterised antibiotics, chloramphenicol, griseofulvin, and penicillin have been found to be readily translocated; streptomycin, aureomycin, and terramycin are translocated in some plants but not in others; gramicidin, neomycin, pyocyanin, and subtilin are not translocated. The data on translocation of antibiotics have recently been reviewed by Crowdy & Pramer[16] and they conclude that neutral and acidic antibiotics are most readily translocated.

Effective disease control has been reported following the use of streptomycin, aureomycin, penicillin, and griseofulvin, considering only well-characterised antibiotics. You will perhaps gather from this list that most of those antibiotics so far tested have been produced in the first place for control of human or animal diseases. These will not necessarily be the best for plant disease control. I think that selection of antibiotics for control of specific plant diseases may finally offer the best results. This approach is being adopted by Leben, Keitt, and their collaborators at Wisconsin[17,18,19,20,21] and by Krassilnikov and his collaborators in Russia.[22,23,24] The latter group have isolated an antibiotic ("No. 16") from a species of Streptomyces, which shows promise for control of bacterial diseases of stone fruits and of black arm of cotton.

All the antibiotics I have mentioned, with one exception, are antibacterial but with little or no effect on fungi. The one exception is griseofulvin, which, as we have previously reported[25] is taken up by the roots of many plants and effectively controls Alternaria solani on tomato, Botrytis cinerea on lettuce, and Erysiphe graminis on oats and barley. I shall have a little more to say about this substance later.

There was no a priori grounds for expecting antibiotics to be readily translocated in plants. The fact that so high a proportion of those tested have been found to be translocated encourages one to suppose that many non-phytotoxic compounds will behave similarly. The antibiotics are worth further attention because there does seem to be a reasonable prospect that among them will be found substances which exhibit that specificity of action which is desirable in a systemic fungicide.

OTHER SYSTEMIC FUNGICIDES

I do not propose to deal in any detail with other substances which have

been shown to control plant disease systemically. I want to make the point that the three groups of substances I have just dealt with are certainly not the only ones which will be found effective. Among synthetic compounds which do not fall into these groups, but which show undoubted activity, I may mention 8-hydroxyquinoline, certain benzthiazole derivatives[26] and benzoic acid.[27]

TRANSLOCATION AND STABILITY IN THE PLANT

There has still been little work reported on translocation and stability of systemic fungicides or bactericides in the plant but I should like to say a little on this aspect. I am indebted to my colleagues Dr. Crowdy, Mr. Grove, Mr. Gardner, and Dr. Pramer for permission to quote from work as yet unpublished.

In our early work on griseofulvin[25] we demonstrated translocation in wheat seedlings by adding the antibiotic to the culture solution in which the plants were growing and by showing that after an interval of a few days griseofulvin could be detected by bioassay in the guttation drops. Our bioassay is based on a characteristic distortion of the germ-tubes of Botrytis allii in a spore germination test. It has been suggested to me that such a bioassay must be far from specific and that consequently our results could not be considered to afford conclusive proof of translocation of griseofulvin unmodified or that it was griseofulvin itself which was protecting the leaves from fungal attack. To that I would reply that we carried out a systematic investigation of the bioassay and are quite confident that only griseofulvin, and a restricted number of substances derived from griseofulvin by minor modifications of the molecule, produce the characteristic response. We have even weightier evidence that griseofulvin reaches the leaf unchanged and I shall describe this later.

Using this guttation drop bioassay technique my colleague Miss Stokes[28] has investigated in a preliminary way some of the factors affecting uptake and translocation of griseofulvin in wheat seedlings. She has arrived at the following conclusions:-

(a) the amount of griseofulvin entering the plant is related to the concentration applied in the culture solution, within the rather restricted range enforced by its low water-solubility;

(b) conditions favouring rapid transpiration, such as increased temperature, low humidity or air movement, increase the uptake and translocation of griseofulvin; conversely, at low temperatures or in conditions of high humidity, uptake is minimal;

(c) there is evidence of accumulation of griseofulvin in the leaves, so that concentrations appearing in guttation drops may exceed that present in the culture solution in which the plants are growing;

(d) uptake is inhibited by such respiratory inhibitors as sodium azide, phenylurethane, and 2:4-dinitrophenol, but not by sodium fluoride; concentrations of the inhibitors can be found which inhibit uptake of griseofulvin without obviously affecting growth of the plant or the amount of guttation.

I would not wish to speculate too freely on the practical significance of these preliminary results, but at least they establish the point that if a systemic fungicide is introduced into a plant from the root system, the rate

of uptake and translocation is likely to be markedly affected by environmental change. It means, too, that if in experimental work one wishes to standardise the dose applied, great care must be taken to standardise environmental conditions.

Crowdy & Pramer[29] have followed griseofulvin uptake in broad bean plants by bioassay of expressed sap and of water and chloroform extracts of plant parts. They find that by chloroform extraction far greater quantities of griseofulvin can be found than by the other two methods. They tentatively conclude that griseofulvin is present in two forms, "free griseofulvin" extractable in water, or present in expressed sap, and "bound griseofulvin" not extractable by these procedures, but extractable with chloroform. This interesting observation merits further study. My colleagues Crowdy, Gardner, & Grove (unpublished results) have succeeded in isolating pure griseofulvin in crystalline form from leaves of plants supplied with griseofulvin in the culture solution, thus confirming the results obtained by bioassay. Crowdy (unpublished results) has also shown that griseofulvin is not stable in the broad bean plant, but that it is gradually lost, only 50 per cent. of an initial dose being recovered after 5 days.

I should like to turn next to the antibiotics chloramphenicol and streptomycin. Dr. Pramer[30] has studied the translocation of these two substances in broad bean plants, supplied with the antibiotic through the roots in a culture solution. The amount of chloramphenicol found in the plants varied directly with the concentration in the culture solution, where the concentration in that solution was varied in the range 10-50 μg./ml. The antibiotic was found throughout the plant after only 19 hr. treatment with a 200 μg./ml. solution, but the concentration in the base of the plant was greater than at the top. If the treatment was continued for 5 days the concentration of antibiotic became even throughout the plant. If, after treatment, the plants were grown on in a solution free of antibiotic for a further 5 days, the upper leaves were found to contain more chloramphenicol than the lower leaves. The accumulation of chloramphenicol was a linear function of water uptake. These results were based on bioassay, but pure chloramphenicol was isolated from leaves by chemical means (Gardner, Grove, & Pramer, unpublished results).

Streptomycin behaved very differently. Using rooted broad bean plants in the same way as the experiments I have just described, very high concentrations of streptomycin had to be supplied in the culture solution (ca. 500 μg./ml.) before any could be found in the plant, and then it could only be found in the base of the plant. On the other hand, streptomycin was taken up more readily by tomato plants, and cucumber plants[31] actually took up streptomycin more readily than chloromycetin.

You will gather from these results that it would be most dangerous to try to generalize about the translocation of systemic fungicides and bactericides. The process differs in different plant species and different compounds behave differently. A great deal more information will be required before our knowledge of the behaviour of these materials can be considered to be on a firm basis. You will gather too that it may not prove easy in practice to ensure the most favourable distribution within the plant of a systemic fungicide or bactericide.

All these results refer to application through the roots. It may well be that in practice application through the foliage will be more attractive, but

as yet little experimental work has been done on this method of application.

SOME SPECULATIONS ON THE PRACTICAL USE OF SYSTEMIC FUNGICIDES AND BACTERICIDES

I will conclude by dealing in a rather speculative manner with three questions that are often asked concerning the practical use of systemic treatments. I emphasise the speculative nature of my remarks because practical experience is at present negligible.

How will systemic fungicides and bactericides be best applied?

Two routes need to be considered. Application through the roots has many disadvantages under normal circumstances. Losses by microbiological degradation, or adsorption on certain constituents of the soil would inevitably be considerable. Only plants grown in water or sand culture could be effectively treated by this route. In the normal circumstances of field culture application through the foliage would appear to be a preferable route. Some known systemics, including those of the growth regulator type, the sulphonamides and some of the antibiotics do appear to enter the plant when applied in this way, but little is known of their subsequent translocation under such circumstances.

What are the advantages of systemic treatment over conventional surface treatment?

Many advantages can be suggested, but few have been confirmed. Results of experimental work seem to confirm the view that uneven application might be compensated for. It seems likely, but there is no experimental evidence that I know bearing on the point, that under conditions of heavy rainfall losses by leaching might be less with a systemic treatment than with a superficial one, but against this possible advantage one has to set the probable loss of material as a result of metabolic activity of the plant being treated. The theoretical possibility of control of established systemic infections and of root diseases is at present a theoretical possibility only, and little if any experimental facts are available that would enable one to evaluate it.

What would be the cost of systemic treatment?

An effective and safe systemic fungicide or bactericide has to meet nearly as many exacting requirements as a drug for human chemotherapy and so is likely to be costly. Certainly few if any of the substances at present showing promise for systemic treatment are of the same order of cost as conventional fungicides or bactericides. It is really far too soon to begin estimating costs since use of an expensive product might be justified if its superiority over conventional treatments were sufficiently great.

CONCLUSION

The evolution of systemic fungicides and bactericides is in many respects proceeding in a reverse direction as compared with the evolution of conventional fungicides. In the latter case practically efficacious products were found by empirical means, and theories of modes of action followed at a considerably later date. In the case of the systemics there seems to be a marked tendency for theory to precede practice. This may be logically

admirable, but judged by the papers already published it would appear that more work in the field is required and we await such results with interest."

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Discussion: DR. LUCAS asked if there was any evidence of plant pathogens becoming resistant to antibiotic action.

DR. BRIAN stated that it was possible in the laboratory to produce resistant strains, but he did not think there was reason to expect that possibility in the field with fungi.

DR. KEYWORTH stated that it was known that many plants possessed a defensive mechanism and he asked whether Dr. Brian had considered the possibility of injecting stimulants, which would cause the production in the plant of substances inhibitory to pathogens.

DR. BRIAN replied that very little was known about the origin and mode

of resistance in plants to pathogens and that the process was intimately related to metabolic activity. Until more was known about the latter it was difficult to commence investigations into this specific subject.

Subject: Low volume concentrate spraying of fruit trees

by M. H. MOORE
(East Malling Horticultural Research Station)

DR. MOORE said that he would deal with only one aspect of low volume spraying, a subject which was being investigated in many places. The delegates would have an opportunity of seeing some of the work in progress at East Malling during the Conference. He was mainly concerned with the use of fungicides for the control of certain diseases, more especially scab (Venturia inaequalis), and powdery mildew (Podosphaera leucotricha) on apples, and he was collaborating with engineers, particularly at the National Institute of Agricultural Engineering.

Sprays of high concentration and low volume were originally employed for plant disease control because of the crude machinery with low mobility in use at that time. As the speed of spraying was accelerated by the introduction of improved machinery greater volumes and higher pressures gave improved results, until volumes became so great that problems were created in water supply and transport of spray fluids. A point had now been reached where excessive volumes of liquid were being used to disperse small quantities of chemicals. The trend was now to turn deliberately to the use of low volume concentrate with machinery designed to give adequate dispersal, and thereby to avoid the injury to plants associated with the earlier crude methods.

The work he was going to describe was commenced with the standard fungicide lime-sulphur, which controlled red spider, mildew, and scab, and about which a good deal was known. Their principal problem was to apply concentrated lime-sulphur in very small droplets with a sufficiently good cover to give adequate control without spray damage. Their first attempts were encouraging and it was decided that such a spray was safe to use. They had previously tried dusts, but they were found to be non-persistent, which was not the case with lime-sulphur.

DR. MOORE went on to say that being assured of the safety of this method they next investigated the fungicidal efficiency of the concentrate, and found that provided the droplet size was small enough (40 to 50 μ) and distribution satisfactory the control obtained of scab and mildew was equal to that of the conventional hydraulic method.

An excessive amount of spray deposit, a result of the fixed nozzle they were using, however, caused severe injury to the trees. Eventually an adjustable paint gun was adapted in which both the supply of air and liquid could be controlled. This was found to be very satisfactory for spraying the small experimental trees (Cox's Orange Pippin, about head high). The experiments were designed to ascertain the most satisfactory concentration and volume to give adequate control of disease without damage. Droplet size and distribution were assessed by an examination of ordinary 3 in. glass slides suspended from the branches of the trees during spraying. It was found that the best balance between control and spray damage for that type of

tree was an application of about 3 gals. of concentrate per acre, compared with 180 gals. of hydraulic spray.

Following up these findings and by varying the concentration of lime-sulphur it was found that 81 per cent. gave better control of scab, mildew, and red spider with less fruit russetting and improved foliage than any of the lower concentrations, when the more caustic spray might have been expected to increase fruit injury. DR. MOORE concluded from this that russetting was not a direct spray effect, but was caused by weathering and that the improved foliage, obtained by control of red spider, protected the fruit and reduced skin blemishes.

He continued that in his opinion the results obtained so far justified in principle the acceptance of very low volume spraying with high concentration but this did not necessarily imply its commercial success.

In collaboration with the National Institute of Agricultural Engineering a small prototype atomising spray machine had been constructed and would be demonstrated to delegates on their visit to East Malling where preliminary trials had been conducted. He said that the main problem now was the correct method of application of atomised concentrates, for which the employment of a skilled operator might be a necessity. This probably would not be popular with growers who have to consider farm economics, but it would be interesting to see the two methods compared when further refinements had been made to the spraying machine.

Discussion: DR. WELSH (Canada) asked whether any means had been found of combining insecticides in powder form with liquid fungicides.

DR. MOORE replied that DDT had been applied to 50 per cent. lime-sulphur, whilst nicotine and other chemicals were being tried, but they were all in liquid and not powder form.

DR. KEARNS (Long Ashton) said that in the West Country high winds had to be contended with, which made the use of very low volume sprays impossible. It had been realized that there was great waste with the run-off washes, and the aim now was to apply high speed sprays of normal concentration.

DR. MARSH (Long Ashton) added that Dr. Kearns implied the use of, say, 1 per cent. lime-sulphur, at 80 gals. instead of 300 or 400 gals. per acre.

Paper: Control of black pod disease (Phytophthora palmivora) of Cacao by fungicide in the Western region of Nigeria.

by C. A. THOROLD
(Department of Agriculture, Nigeria)
[Presented by J. M. Waterston, Nigeria]

DR. WATERSTON in introducing the paper said that at the last Commonwealth Mycological Conference the economic difficulties of using fungicides on peasant crops in the tropics had been stressed. The recent high price of cocoa, however, had caused these views to be modified and had prompted an investigation into the economics of black pod control by fungicides.

He wished to emphasize that the culture of cacao in West Africa was haphazard and primitive, and it was noteworthy therefore that the cocoa farmer had taken an enthusiastic interest in the spraying experiments; but the success obtained had brought him increased revenue, which was expressed by improved housing and other domestic possessions.

He stated that the present experiments were only preliminary and more fundamental studies were required on the pathogen and the machinery.

INTRODUCTION

Spraying experiments carried out on cacao in Trinidad (10) showed a reduction in number of diseased pods (including witches' broom pods and other pod diseases) by monthly applications of Bordeaux mixture. When numbers of diseased pods were expressed as a percentage of the total (healthy plus diseased) pods the reduction was from 33 per cent. on untreated trees to 19 per cent. on treated trees. Better results were obtained in Nigeria (10), where percentages ranged from 58 to 74 on untreated trees, and between 3 and 10 on treated trees. Pod wilt (Baker & McKee (2)) accounted for some of the pod losses on both treated and untreated trees in Trinidad, and it is likely that pod wilt occurred also in Nigeria. It will be shown later that the incidence of wilt, as affecting pods and cherelles (3 and 4),needs to be considered in connexion with total pod counts.

Conventional Bordeaux mixture was used in the Trinidad experiments, but in Nigeria 'carbide Bordeaux' was employed on account of lack of lime, as explained elsewhere (10). The preparation (1 lb. copper sulphate, 6 oz. calcium carbide, in 10 gals. water) leaves a deposit both conspicuous and tenacious.

Plot experiments in Nigeria, using about 200 trees, in 1950-51 were extended to about 220 acres treated in 1953-54 in order to determine the economics of spraying and to gain experience in organizing the work on a field scale. Farmers have been favourably impressed by the degree of black pod control obtained, and two started spraying on their own initiative, whilst others asked for pumps and materials, which were not then commercially available in Nigeria. It was difficult to resist this demand and in order to prevent indiscriminate spraying, possibly done inefficiently with unsatisfactory equipment and materials, arrangements were made for the financing of a scheme to provide 2000 Four Oaks Kent Pattern (4 gall.) spray pumps together with chemicals and ancillary equipment for the 1954-55 season, to be purchased by farmers on agreed terms. The farm must be high-yielding (average of 12 or more pods per tree) and in an area of bad black pod disease (about 50 per cent. loss), otherwise frequent removal of affected pods is recommended. The spraying programme proper starts in June, when applications are given at intervals of three weeks until November or December, when spraying ceases. Advisory officers of the Department of Agriculture inspect the farms and assist the farmers. In the second and subsequent seasons spraying will be at six-week intervals, following the first year's application at three-week intervals.

This spray schedule has been found to keep black pod losses below 10 per cent.

Results from farmers' treatments of their farms are not yet available. In the meantime the object of this paper is to present information on the

following aspects of black pod control:-

(i) spraying costs, (ii) methods of assessing results in field-scale work, and (iii) consideration of possible effects of spraying on total pod production.

SPRAYING COSTS

Records have been kept of the materials and labour employed in field-scale spraying trials. Results have already been given for 1952-53 with respect to the treatment of 40 acres (11). Further information is now available for this area, which received infrequent applications (six-week intervals) in 1953-54; during the same season, an area of about 185 acres received frequent applications (three-week intervals).

The total cost per acre for spraying the 40 acre plot was £17.6 for the first year (frequent spraying) and £10·9 for the second year (infrequent spraying). The cost of spraying the 185 acre plot was £14·1 for the first year. It can be shown that the above costs are economic under the specified conditions (average yield of 12 pods per tree and 50 per cent. black pod loss, without spraying) and when allowance is made for cocoa which would be obtained without spraying. Harvesting and preparation costs are not considered. It is appreciated that they will be somewhat greater when farms are sprayed, because there will be additional pods to be reaped and extra cocoa to be fermented and dried. Under conditions in Nigeria it may be assumed that 12 pods are equivalent to 1 lb. dry cocoa. At 600 trees per acre averaging 12 pods per tree, the expected yield is 600 lb. dry cocoa.

In the area where these experiments are sited the yield in unsprayed plots is only 300 lbs. per acre valued at £22. When the frequent and infrequent spray schedule is followed, the yields are increased to 540 lbs. dry cocoa per acre, and the gross return from the 40 acre experiment, after deducting the cost of spraying is £23·4 (frequent applications) and £30·1 (infrequent applications). This represents a profit from srpaying of £1·4 and £8·1 per acre, respectively. The figures for the first season spraying (frequent application) for the 185 acre experiment are a gross return (after deducting costs) of £26·9 representing a profit from spraying of £4·9 per acre.

The spraying of cacao yielding fewer than 12 pods per tree, may also be remunerative, but until farmers gain experience in the work, it is desirable to restrict the scheme to make sure there is no disappointment through inadequate returns being obtained.

ASSESSMENT OF YIELDS IN LARGE-SCALE EXPERIMENTS

When trials progressed beyond the plot scale the problem of sampling arose owing to variability resulting from different treatments and plantings on individual farms (Pearce & Thom (17)).

SAMPLING PROCEDURE: One hundred bearing trees (as 50 pairs) were selected, and one of each of the 50 pairs allocated at random to receive either spray or no treatment for each of the frequent and infrequent application areas. Accordingly, there were 50 treated and 50 untreated trees for each of the two treatments. The results are summarized in Table 1.

Table 1. Mean numbers per tree of black pods (B. P.), healthy pods (H. P.), and total pods (T. P.), for trees receiving frequent and infrequent applications, respectively.

	B. P.	H. P.	T. P.
Frequent applications	0.9	11.0	11.9
No treatment	9.5	7.9	17.4
Infrequent applications	0.0	9.7	9.7
No treatment	2.1	10.0	12.1

For the purposes of analysis, the number of total pods per tree (X) was transformed (log (X + 1)), when it was found that the mean value was significantly less in the case of the frequently sprayed trees as compared with untreated trees, but the difference was not significant in the case of infrequent spraying, as shown below:-

Table 2. Transformed value of total pods per tree

	Transformed value of T. P. (mean)	Difference
Frequent applications	1.02	0.15*
No treatment	1.17	
Infrequent applications	0.91	0.09
No treatment	1.00	

*Significant at 5 per cent. level.

A depressing effect on perennial yield has also been reported from the Ivory Coast by Renaud (8). He supposes that the apparent decline in number of total pods attributed to spray applications is due to action on the flowers either directly or indirectly through a repellant effect of the Bordeaux mixture on pollinating insects. There was apparently no direct evidence in support of these suggestions. In the course of spraying experiments in Nigeria, it has not been possible to detect any direct effect of spraying on flower setting.

DR. WATERSTON interpolated "Renaud comments that the 5 per cent. concentration of copper sulphate was stronger than that commonly used, and that 'burning' of the leaves was not observed, but no reference is made to 'burning' of flowers having been actually observed. Thorold has not employed Bordeaux mixture or carbide Bordeaux at concentrations other than 1 per cent. by weight of copper sulphate in water (assuming one gall. water weighs 10 lb.). In view of the fact that cacao trees produce flowers during the greater part of the year it is not to be expected that there would be any marked effect on total pod production even though a single application of 4 per cent. or 5 per cent. Bordeaux mixture had affected flowers open at time of spraying. There was no apparent effect on total pod production in the course of weekly applications of 1 per cent. carbide Bordeaux during two

seasons in Nigeria. Thorold believes that some other explanation must be sought."

In making comparisons between total pods for sprayed and untreated trees, respectively, it is assumed that the groups of pods recorded are directly comparable. There is reason to believe that this hypothesis is not entirely justified. Phytophthora palmivora can infect fruits of all ages and instances have been observed when this fungus has apparently invaded fruits which are already wilting. It seems that discrepancies in total pod numbers for sprayed and untreated trees would receive at least partial explanation if the term 'black fruit' was substituted for 'black pod' disease. Comparisons should then be between total fruits rather than between total pods. Fruits would then comprise the following categories:- healthy and diseased mature fruits; wilted fruits and all 'black fruits'. It is likely that wilted cherelles will be disregarded when present on sprayed trees, but if such cherelles had not been sprayed they might have been infected by P. palmivora and would then be included as 'black pods' in total 'pod' counts. Unfortunately, it is not possible to verify this supposition directly. However, when all fruits present on trees at a particular occasion are counted the numbers of such 'total fruits' are not markedly dissimilar on sprayed and untreated trees.

It is inevitable that immature fruits are removed as black pods in the course of recording disease incidence, because black pods allowed to remain on the trees would have a secondary effect. It seems that the removal of immature cacao fruits may be comparable to the procedure of 'thinning' temperate fruits, beneficially affecting both size and ultimate numbers (9). Unfortunately the analysis of observations in such studies presents inconsistencies (5 and 6). The writer is not aware of any study of the effects of 'fruit thinning' of cacao, but certain observations have been made which appear to be relevant.

With the object of gaining information on the possibility of crop-forecasting the total fruits (x) on 4,936 individual trees at Agodi Cocoa Farm (11) in June 1952 and the total pods (y) in the 1952-53 season were recorded. The regression coefficient (b_{xy} = 1·23 ± 0·202) is positive and highly significant.

It seemed of interest to ascertain whether there was any difference between total pod numbers for two populations of trees, one with no black pods removed, and the other with large numbers removed. Accordingly, two samples were selected systematically, one sample of 25 individuals contained trees which had 20 or more total fruits in June and 10 or more black pods in the course of the season, the other sample of 25 individuals contained trees which had 20 or more total fruits in June and no black pods in the course of the season. The results showed that whereas the two samples selected may be regarded as comparable with respect to the mean number of fruits in June, sample (I), in which relatively large numbers of black pods were removed (17.24 per tree), bore an ultimate number of total pods (59·80) greatly in excess of the number of total pods (27·04) in the other sample (II), in which no black pods were observed. These results may be interpreted as indicating that the removal of black pods has accounted for an increased number of total pods being produced. If this comparison is valid and the interpretation correct, an explanation would be provided for discrepancies between sprayed and untreated trees with regard to number of total pods. Although it is of some fundamental interest to explain such discrepancies, it is considered that they are not to be interpreted as presaging a decline in cocoa yields through the practice of fungicide spraying. Bordeaux mixture has been applied for at least 20 years to cacao trees in Fernando Po for the control of black pod disease; there is no evidence that cocoa yields have declined there during this

period. According to this explanation the discrepancies may be an 'artefact arising through a stimulation of fruit production due to removal of immature fruits as black pods. It is probable that numbers of total pods recorded for untreated trees may include immature wilting fruits infected by P. palmivora but which would in the absence of infection have been disregarded, as for instance on sprayed trees.

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Paper: The control of black pod and witches' broom diseases in Trinidad.

by PAUL HOLLIDAY
(Imperial College of Tropical Agriculture)
[Presented by D. Rhind, Colonial Dependencies]

Black pod (Phytophthora palmivora) and witches' broom disease (Marasmius perniciosus) are the most important fungus diseases of cacao in

Trinidad. Losses due to each are difficult to separate because their pod symptoms are liable to be confused. All the beans in pods infected by witches' broom are usually destroyed and therefore the number of infected pods gives a fair indication of losses. Losses from black pod disease have generally been thought to be due to infection of mature or nearly mature fruit. Recently, in Costa Rica, emphasis has been laid on infection of the young pods (cherelles) but in Trinidad this factor is probably not important. A high percentage of cherelles wilt anyway whether they have been infected or not. Good beans are often recoverable from fruit attacked by black pod disease, and therefore bean weights are the only reliable method of determining losses. In the relatively drier parts witches' broom disease is the more important but in areas with a rainfall, over 100 ins., per annum black pod can be more severe. The dry season (February to May) has an important limiting effect on both diseases. The rains begin in June but it is not until October that pod rots become serious, remaining so until March. This period (October to March) occurs at the time when the main crop is being reaped.

WITCHES' BROOM can be controlled by the removal of brooms. The life cycle of the fungus, from infection to spore formation, takes three to four months but in Trinidad it is not necessary to remove brooms from the trees at intervals as frequent as this. The reason is the existence of a dry season which prevents the growth of sporophores at this time of the year. As a result two removals each year, at specific times, are considered sufficient. This method of control can work well under certain conditions. If trees are too tall, badly shaped, and irregularly spaced control is less efficient than if they are kept well pruned in regular stands. Losses can be kept to 10 per cent. and even less, if local, partially resistant, clones are planted.

Spraying against witches' broom disease has been inconclusive until recently. Although, lately, it has been shown that Bordeaux mixture will reduce the incidence of fan brooms the difficulty of spraying whole trees under present conditions is a real one. More trials are needed and it is doubtful whether such trials are justified at present. But spraying the cropping regions of the trees only — an operation made simple by the cauligerous habit of cacao — is worth consideration. The heaviest losses occur between October and March; during the remainder of the year they are insignificant. Spraying should begin in early July to protect pods reaped in October because diseased pods recorded in the field are infected three months earlier. From experiments begun in 1950 we have found that copper sprays applied once each month from July to the end of the year reduced losses between October and March by amounts varying from 36 to 70 per cent. Bordeaux mixture is slightly better than cuprous oxide (perenox) but the latter fungicide together with copper oxychloride (blitox) gave good control and are worth further trial. In 1952 some other fungicides were tested. Lime-sulphur (sulfinette) gave as good control as the proprietary copper compounds but zinc dimethyl dithiocarbamate (ziram) and tetramethyl thiuram disulphide (fernide) were disappointing.

Host resistance is likely to be the best method of control of witches' broom disease. Local selections made by the Imperial College of Tropical Agriculture (ICS clones) are variable in their reactions to it. Some are much less susceptible, significantly so, than others so that in selecting clones for commercial plantings, consideration is given to this factor. Fortunately, clones which show resistance to pod-infection usually show comparable

resistance to infection of the young shoots suggesting that the resistance factor in the Trinidad population is not a simple morphological one. Resistance, approaching immunity in effect, has been shown in two trees (known as SCA 6 and SCA 12) which originated as seedlings brought from the upper Amazon valley. In the field no brooms have ever been recorded on them and attempts to infect cuttings of SCA 6 and 12 and produce brooms artificially failed. Pods of these two trees are very occasionally attacked but the percentage infected is so slight as to be insignificant. Inoculation techniques previously used with M. perniciosus gave only a low percentage of success and studies on the inheritance of resistance were therefore handicapped. However, a method in which seedlings are inoculated four days after germination gives 100 per cent. success. Selfed seedlings from a fairly susceptible Trinidad clone (ICS 1 (Imperial College Selection 1), when inoculated by this method, show very severe symptoms and 79 per cent. die. The remainder show very poor, stunted growth. The clones SCA 6 and 12 will not set seed with their own pollen but both will cross with ICS 1. In the cross SCA 6 × ICS 1 none of the seedlings inoculated at four days die and, whilst all show symptoms, only 6 per cent. approach those on ICS 1 seedlings in severity. In the other cross SCA 12 × ICS 1, 10 per cent. of the seedlings die and 24 per cent. show severe symptoms. This test therefore confirms that both SCA 6 and 12 are highly resistant, the former clone being the more so. It seems probable that some prediction about the behaviour of seedlings, which have been tested by this method, towards the disease in the field can be made. Field trials are in progress to test this. We do not yet know how widely applicable this inoculation method will be; whether, besides revealing resistance in Amazon clones, it will also enable us to assess resistance in local material.

BLACK POD disease has been largely ignored since 1928 when the then more obviously serious problem raised by witches' broom arose. It also is strictly seasonal in its incidence and only becomes serious in the latter part of the wet season from October to January. The form of the disease on the young shoots known as "chupon blight or wilt", so prevalent in Costa Rica, is absent here; this is probably due to the relatively drier conditions prevailing in Trinidad. In the wetter eastern regions black pod may be more serious than is often thought because losses due to it have been attributed to witches' broom. Under the Trinidad plantation system of generally large holdings fairly frequent harvesting has been possible. Picking at intervals of about four weeks enables good beans to be reaped from infected pods, pods which would have been lost if harvested at less frequent intervals.

Treatment by fungicides to control black pod has never been regularly practised. It is unlikely that it would be worth-while, unless witches' broom could be controlled at the same time, except in areas where the rainfall amounts to 100 ins. or more per annum. In one experiment (1952-53) in such a wet area — but designed primarily to investigate witches' broom control - perenox and blitox both gave significant control of black pod. Five applications (blitox) at monthly intervals from July to November reduced infection during October to January from 36 per cent. to 10 per cent. If the spraying had been done later in the year there is little doubt that control would have been better. The other fungicides mentioned under witches' broom disease were ineffective against black pod.

No conclusive demonstration of resistance to P. palmivora in cacao has yet been made although there are indications that pod resistance does occur. Inoculation technique is simpler than is the case with M. perniciosus. The

fungus sporulates readily in pure culture and pure zoospore suspensions can be obtained. Sporangia do not produce zoospores until temperatures reach about 20°C. In Trinidad this does not occur until night falls but if cultures are incubated at this temperature at any time during the day zoospores are easily obtained. Filtration through sintered glass of an appropriate porosity ensures a pure suspension. Artificial infection is simply and quickly produced by applying zoospores under 100 per cent. humidity conditions to leaves or pods of any age. The development of resistance tests is however handicapped by lack of knowledge of the fungus itself. We know that different strains occur on different hosts but are ignorant of the differentiation of strains on a single host. If several strains occur on cacao in Trinidad some are likely to be more important in the spread of the disease than others. What, too, is the relative importance of the various spore forms?

The losses due to both diseases could be lessened if the measures now known to be effective were more widely and more efficiently applied. In the case of witches' broom a careful attempt must be made every April and October to remove as many brooms as possible; the disease may then be ignored, as far as this method is concerned, for the remainder of the year. As to black pod attention to more regular and frequent harvesting during the wet season is wanted. Because of tree heterogeneity spraying in existing old fields is likely to be unsatisfactory and further yields from such cacao would not justify this method of control. But there are several factors which warrant further spraying trials. More and more fields of clonal cacao are coming into bearing and they will yield better than existing seedling fields. The more regular planting in these young fields would make operations simpler and cheaper. Prices are now much better than in the past. Whilst it may not be worth while to spray against either disease alone it would be possible to spray against both simultaneously since they are serious at a similar time of year, and since copper is effective in both cases. Resistance studies are most important and have been the most neglected where black pod is concerned. This is surprising considering it is more widespread and results in higher losses. In the development of breeding schemes the opportunity to incorporate material resistant to both these fungi in future commercial varieties must not be lost, even to some extent in those areas where either disease may, at present, be absent or unimportant.

Discussion: MR. RHIND said that the absence of breeding referred to by Mr. Holliday was explained by the absence of resistant material. A single tree in Samoa known as Laffia No. 7, however, had remained free from black pod for a long period and although first attempts to transport material had failed there were great hopes that it would eventually form a valuable parent for breeding in West Africa and Trinidad.

MR. OWEN (Reading University) said that his experience on the Gold Coast with similar experiments led him to believe that better control of black pod can be obtained by weekly harvesting, and that spraying was only economically practicable when cocoa prices were abnormally high. He felt that propaganda should be extended on those lines amongst the West African farmers in preparation for a slump in cocoa prices. He had also found a problem in obtaining clean water and suggested that consideration should be given to low volume spraying if spraying were to be employed at all.

MR. DADE (C. M. I.) confirmed the efficacy of frequent harvesting but explained the difficulties of persuading peasant farmers to adopt this method. He maintained that estimated crop losses were often misleading,

instancing experiments which he himself had conducted on the Gold Coast, whereby official estimates of 50 per cent. loss were shown in fact to be no more than 10 per cent. It was not possible to make snap judgments of disease losses, one of several reasons being that there is a fairly high natural shedding of 'cherelles' by cacao. He supported Mr. Owen in his remarks about the difficulty of water supply and was in favour of investigating the use of low volume concentrates. He felt that the difficulties of black pod control during periods of low cocoa prices was as yet by no means solved.

DR. WATERSTON replied that there really was no water problem as it was only necessary to spray during the wet season when water was plentiful. Regarding the figure of 50 per cent. disease this applied to a selected area where the loss had been accurately assessed at an average of 50 per cent. year after year. He agreed that frequent harvesting would more or less eradicate the disease but this was a council of perfection as the farmers would not do it, and unless a co-operative fermentary were established individual pickings were insufficient in quantity to ferment properly. He realised that the behaviour of Phytophthora palmivora varied in different countries, and it was interesting that the canker stage had not been observed in Nigeria.

Meeting: Tuesday, 20th July, 2.30 p.m.

Chair: DR. R. S. VASUDEVA

Subject: Miscellaneous contributions

Paper: Tobacco disease problems in Southern Rhodesia

by G.R. BATES
(Southern Rhodesia)

Tobacco is the most important cash crop of Southern Rhodesia and consequently plays a large part in the country's economy. During the last decade the industry has trebled in size and in 1952-53 it included some 2,500 growers who produced more than 105 million pounds of tobacco which realised nearly £17½ m. sterling. This figure represented more than half the gross value of the total agricultural output of the country in that particular year.

The rapid development of the industry has meant the opening up of new areas where soil and climatic conditions often differ from those of the older tobacco growing districts. As might be expected this expansion has led to fresh problems, amongst which those concerned with pests and diseases are not the least important. The industry is still young enough to be receptive of new ideas and techniques. In recent years many new cultural methods have been introduced, some derive from research studies and others stem from the keen spirit of enquiry shown by the growers themselves. For example, growers are now advised to plant much earlier in the season to obtain better stands and yields. It is also claimed that such early plantings have a better chance of escaping the major leaf spotting diseases. Early planting often requires the use of watering to establish the young seedlings. Some farmers have adapted irrigation methods for this purpose and a few are producing crops entirely under irrigation.

Another well known characteristic of the local industry is the extraordinary eagerness of growers to switch to new varieties, usually obtained from other countries, with little or no preliminary testing to determine their general characteristics and disease behaviour.

All these factors might be expected to have a considerable bearing on the disease situation but it is interesting to find that the general pattern regarding the distribution and importance of the major tobacco diseases has remained substantially unaltered over the past decade. However, the influx of new growers, many of whom have little knowledge or experience of the hazards of tobacco culture has undoubtedly been connected with localised outbreaks of disease in recent years.

Before dealing with more specific instances it may be pertinent to review briefly the history of tobacco research and more particularly of tobacco disease investigations in Southern Rhodesia. The first organised studies into tobacco diseases were initiated by my predecessor, Dr. J. C. F. Hopkins, who started his investigations as far back as 1926 and, within a few years, had accummulated sufficient information to publish the first handbook on diseases of tobacco in the Colony. Subsequently, in addition to many original papers on the subject, he produced a valuable supplement to the original handbook. During and immediately after the war period when the industry was expanding so rapidly, the shortage of plant pathologists made it necessary to concentrate mostly on the advisory aspect of tobacco diseases. However, some attention was given to the field control of certain leaf spot diseases, notably frog-eye (Cercospora nicotianae) and brown spot (Alternaria longipes) by means of fungicides applied by modern power driven sprayers.

Tobacco research was first placed on a proper footing by the establishment in 1934 of the Tobacco Research Board and a tobacco research station at Trelawney was opened the following year. The work was seriously interrupted by the war and to meet the pressing demands of the industry for greater attention to their tobacco problems an interim research scheme, largely financed by the industry, was started in 1947. Three years later the Tobacco Research Board was re-organised, the cost of research being met by both industry and Government. Under this latest arrangement a central tobacco research station has been established near Salisbury whilst the original station at Trelawney will, in due course, be relegated to the status of a field station.

It will be appreciated from these remarks that research on tobacco diseases has suffered from frequent staff changes and lack of continuity.

Only a few of the many diseases recorded are of serious consequence to the crop and no accurate assessment of the losses has been made. In other words the order of priority given to the various diseases is entirely my personal opinion.

Frog-eye. Of all the diseases attacking tobacco frog-eye is undoubtedly the most widespread and probably causes the greatest loss by reducing the value of the leaf. Unfortunately the economic importance of frog-eye is often discounted by growers since on the auction sale floors the presence of a small amount of frog-eye is regarded by the buyers as a sign of well matured leaf and such spotted leaf usually realises good prices. Unless the disease becomes really serious throughout a crop the average grower is

inclined to ignore its presence. The biology of C. nicotianae has not been closely studied and our knowledge of the fungus is rather fragmentary. The spores are wind-borne and, as a general rule, the disease tends to become more severe as the season progresses. Frog-eye is capable of attacking tobacco at all stages of growth consequently great care must be taken to prevent infection developing in the seed-beds. Satisfactory protection is afforded by the standard copper fungicides (Bordeaux and Burgundy mixtures, cuprous oxide, copper oxychloride, etc.) provided they are properly applied at sufficiently frequent intervals. No really economic method of field control has yet been devised and further research is needed on this phase of the disease. The commercial varieties of flue-cured tobacco appear equally susceptible to frog-eye but it seems reasonable to suggest that other species of Nicotiana should be studied with a view to discovering possible sources of resistance. Field observations indicate that plants deficient in nitrogen may be more susceptible to the disease. More critical investigations are therefore desirable regarding the nutrition of tobacco and the incidence of frog-eye.

The same fungus is also responsible for the condition known as barn spot which only develops during the early stages of the curing process. Barn spot can be checked though not entirely controlled by simply raising the initial curing temperature to 100°F. instead of 85°-90°F. as usually practiced. Naturally, increased moisture must also be provided to maintain the relative humidity as near 100 per cent. as possible. Recent observations suggest that the amount of barn spot developing under favourable conditions is inversely related to the rate of nitrogen fertiliser applied.

Brown spot. (Alternaria longipes) has been more intensively studied than frog-eye, probably because of the more spectacular losses it causes in the field. Since it was first recognised by Hopkins about 1931 severe epiphytotics have occurred from time to time in several of the main tobacco growing areas of Southern Rhodesia. More recently it has been responsible for heavy losses both in Northern Rhodesia and Nyasaland. Essentially it is a disease of mature, well-bodied leaf. It is unknown in seed-beds and on young plants in the field. Under favourable conditions of wet, close weather the disease spreads with great rapidity and can cause total loss of crop. Tobacco grown on virgin or long fallowed soil is rarely attacked severely but the disease is progressively worse amongst crops grown on second- and third-year land. This may be due to the accummulation of diseased residues from previous crops and also to nutritional factors. E. A. Riley has shown the susceptibility of tobacco was favoured by lack of phosphorous and potassium whereas plants grown at high nitrogen, phosphorous, and potassium levels were less susceptible. No important differences in varietal susceptibility to the disease have yet been found. Attention has been given to the possibility of controlling brown spot in the field by fungicides but the problem bristles with difficulties. It is not easy to find a fungicide with sufficiently weather-proof qualities to stand up to the tropical storms commonly encountered during the growing season. Moreover, the growth rate of tobacco foliage is so rapid that frequent applications are necessary to provide efficient coverage and protection. On top of these difficulties the tobacco trade objects very strongly to the presence of any surface deposit on the leaf. Field trials with heavy power driven spray equipment were unsatisfactory when the ground was wet, whilst the machines were difficult to handle without damaging the crop. Hopkins has reported encouraging results from the use of knapsack sprayers operated by teams of labourers but this method hardly lends itself to large acreages.

Mildew. Many years ago when the flue-cured variety known as Hickory Pryor was generally grown mildew or white mould (Erysiphe cichoracearum) was considered the most serious disease of tobacco in Southern Rhodesia. Following the introduction of the smaller, narrow leaved varieties of the Orinoco group, the incidence of mildew declined sharply and for a long time the disease was of minor importance. In recent years, however, it has shown a disturbing increase in many districts even amongst varieties hitherto regarded as fairly resistant. The reasons for this renewed activity are not fully understood but may be associated with climatic conditions, the introduction of more susceptible varieties,and the adoption of new field practices in spacing and fertilizer rates. Control measures are difficult owing to the nature and behaviour of the fungus although promising results from the use of certain organic fungicides have been reported from South Africa. It seems likely that the development of resistant varieties offers the greatest promise of success against this disease.

Tobacco anthracnose (Colletotrichum tabacum) was discovered for the first time in Southern Rhodesia in November, 1953, causing a leaf and stem blight of seedlings. This disease had previously been reported from the Southern Province of Northern Rhodesia where it caused serious damage to seedlings. So far, in Southern Rhodesia, the disease appears to have been confined to seed-beds on one farm. The outbreak in Northern Rhodesia was considerably more extensive and resulted in heavy losses of seedlings over a wide area. I am indebted to Dr. E.A. Riley for much information on the disease in Northern Rhodesia. It was thought at first that anthracnose was restricted to young plants in the seed-bed and that it usually disappeared after transplanting in the field. However cases have now been found of leaf spotting and stem canker on field plants. In such cases the stem canker may be so severe as to cause girdling and eventually kill the transplants. Two distinct types of symptom have been recognised on seedlings, the first being discrete dull green leaf spots, the second consisting of enormous numbers of minute spots with light brown or white centres; the latter appears to be due to the numerous infections developing from spores disseminated from the larger "primary" lesions. The disease can be carried over in the soil, and is thought to be air-borne but not seed-borne. Attempts to control the disease by protective spraying with 4-4-50 Bordeaux mixture were not entirely effective although it was observed that these treatments largely prevented the minute type of spotting ("secondary phase"). Some form of soil disinfection is regarded as necessary in addition to protective spraying to bring the disease under control. Work on this problem is in progress. Preliminary experiments indicate that the dithiocarbamates are likely to be superior to copper and mercury compounds as foliage sprays.

Wildfire (Pseudomonas tabacum) continues to be the most serious bacterial disease of tobacco. It is known to be seed-borne but our studies indicate that treatment with 1 in 1000 silver nitrate effectively controls the disease particularly when subsequent washing with water is omitted. Seed from disease-free plants should, of course, be uncontaminated but there may be undiscovered sources of contamination during handling and storage. Some out-breaks of wildfire both in seed-beds and the field appear quite inexplicable unless, as reported from Kentucky, the organisms can survive in a saprophytic form on the roots of indigenous grasses. This possibility requires investigation locally.

Bacterial or Granville wilt (Pseudomonas solanacearum) was discovered by Wickens in 1936. It reappeared on the same farm on land fallowed for nine years, and again on another farm, some 6 miles away from the first out-

break, in 1946. It developed on apparently virgin land in a region where signs of very old native occupation could be seen. After a lapse of eight years two further cases of bacterial wilt were discovered this year in the same general area, though fully 30 miles apart. No severe damage was caused and most of the affected plants produced a reasonable crop. Definite indications of old native occupation were again observed in one of these latest occurrences, also on apparently virgin land.

The disease is far less agressive and damaging than in the United States; possibly our climatic conditions and the long dry winter have something to do with this. P. solanacearum does not like dry conditions.

It seems that at least two forms of P. solanacearum occur in Southern Rhodesia. A so-called potato strain which, though possessing a wide host range, failed to infect tobacco in field trials. The second or tobacco strain has not yet been tested against other potential hosts in the field, but it does infect weeds such as Amaranthus graecizans in a benign fashion.

Virus diseases: Mosaic. There are no new developments to report. Incorporation of resistance into locally favoured varieties is in progress but has not yet resulted in commercially acceptable types. Attention is more directed towards avoiding infection and transmission in seed-beds and field by appropriate hygienic methods and careful supervision. Some of the new household detergents are reported to be very effective in removing the virus from hands of workers.

Leaf curl. The situation remains satisfactory and the disease unimportant. An obvious danger exists of the disease becoming more widespread as the "close season" period tends to be reduced by earlier planting. To overcome this possibility it has been decided to advance the date when all flue-cured tobacco plants remaining in the field must be destroyed from August 1st to July 1st each year.

Rosette. Signs of a fresh cycle of the disease are apparent. It was prevalent in many areas last season. New growers are not only ignorant of its dangers but often unable to even recognise the disease. Increased incidence is possibly linked with this and the ploughing-in of stalks instead of the former practice of pulling and burning. Last season was very favourable for the vector, which occurred in large numbers in most districts.

Recent outbreaks were characterised by far milder symptoms than hitherto observed. There is almost complete absence of severe rosetting, only slight distortion of apical growing point, and negligible stunting. Enough vein distortion occurs to produce "hooded" effect and some puckering on the leaves. The virus complex may have altered, perhaps the "vein distorting" component as isolated by K. M. Smith has become attenuated. Equally possible, of course, the sap transmissible or mottle virus component may have changed.

Discussion: DR. VASUDEVA said that he understood that frog-eye in tobacco was serious under conditions of low nitrogen which was contrary to his observations in other crops. Regarding the control of leaf curl by the removal of other solanaceous plants, as practised in Southern Rhodesia, this was not possible in India owing to the wide host range of the virus there.

MR. MARSH (Long Ashton) asked whether Cercospora was seed-borne and where infection originated.

DR. BATES replied that he did not think it would be seed-borne in Rhodesia as all seed was treated with silver nitrate before sowing. The actual source of infection from year to year was still a matter of conjecture and had not been fully investigated.

In answer to another question he stated that mosaic was controlled in the lands by putting on separate gangs to handle diseased and healthy plants and by hand-washing at the end of every row.

Paper: Oil palm diseases in Nigeria

by R. A. BULL
(West African Institute for Oil Palm Research)
[Presented by R. A. Allen, W. A. I. F. O. R.]

Although the oil palm suffers little when growing in a wild state, several serious diseases occur in plantations and nurseries. Among mature palms the most important diseases which have to be considered at the present time are nutritional in origin, and they are characterized by symptoms which are generally similar to those seen in other crops growing on deficient soils.

The most serious parasitic disease of plantation palms is vascular wilt disease caused by a strain of Fusarium oxysporum, while in the wild groves Ganoderma trunk rot is of considerable economic importance. In nurseries two leaf diseases, freckle and anthracnose, are assuming increasing importance, and a serious root disease called blast has been under investigation for several years.

Since the deficiency diseases of mature palms occur over wide areas in the palm-growing regions of Nigeria, and on certain soils may well be a limiting factor in oil palm cultivation, they will be considered first. At the moment leaf deficiency symptoms are provisionally classified into three groups: (1) yellowing, (2) orange spottings, and (3) chloroses.

Orange frond. An example of the first group is the disease to which the name orange frond has been given, since it is characterised by a pronounced orange discoloration of the leaves. The first symptom is the appearance of olive green or ochre-coloured areas at some point on the leaflet. These early lesions later become yellow in colour, and as the affected areas increase in size the colour of the leaflet changes from ochre to bright yellow, and then to a deep orange. It is at this stage that the foliage of affected palms assumes the characteristic orange colour, which has led us to term the disease "orange frond". Later still the moribund leaflets become infected by fungi and their appearance changes abruptly. Umber-coloured lesions appear within the orange areas, and these quickly spread until the whole width of the leaflet is affected. After a week or two the infected tissues die, dry out, and roll round the midrib.

The cause of orange frond disease has been investigated by means of leaf injection, and spraying techniques, and by chemical tissue tests, using the Long Ashton methods.

A leaflet-tip injection experiment performed on young orange frond palms of three to five years of age suggested that orange frond is caused by magnesium deficiency, and corroborative evidence of magnesium deficiency was also obtained from a spraying trial and from leaf analyses.

There was, however, no direct correlation between leaf magnesium content and the severity of symptoms. These results suggest that once leaf magnesium has fallen below a certain level and deficiency symptoms have appeared the colour changes seen in the leaf represent a progressive degeneration of the chlorophyll pigments which is not associated with a progressive loss of leaf-magnesium.

The results from these various lines of investigation are being followed up by a fertilizer experiment which was laid down in March 1953 on an area of palms suffering from orange frond disease. The aim of the experiment is to study the effect of soil applications of magnesium sulphate on yield and disease symptoms. Four treatments have been applied as follows:

A. Control; no magnesium sulphate
B. 5 lb. magnesium sulphate per palm
C. $7\frac{1}{2}$ lb. of magnesium sulphate per palm
D. 10 lb. of magnesium sulphate per palm

During the period March 1953 to 1954 there was a considerable improvement in the general health of the palms receiving magnesium fertilizer but no improvement in the control plots. The speed of recovery appears to follow the dosage of magensium sulphate applied.

No increases in yield have yet appeared in any of the treated plots. This is not surprising in view of the long period between initiation and maturation of oil palm bunches, and experience from other fertilizer experiments suggests that effects on yield are not likely to appear until some 18 months after the magnesium sulphate was applied.

Orange spottings. We will now consider the orange spotting diseases which are encountered, in varying degrees of severity, almost everywhere that oil palms are grown. In these diseases the yellowing does not spread over the whole surface of the leaflet but is restricted to certain areas of the lamina, so that the latter becomes covered with a mass of orange spots. Ultimately the margins of the leaflets die and drop off leaving a ragged edge, and the leaflet tissue also dies back from the tip. [A detailed description of symptoms was given in the demonstration.]

Diagnoses may be complicated by the appearance of orange spotting and orange frond on the same leaflet, when the development of the two diseases is atypical.

When transverse sections from healthy leaflets and from leaflets showing orange frond or orange spotting are compared the colour and distribution of the chloroplasts in the palisade and mesophyll tissues of healthy and deficient leaves are found to differ considerably. In healthy palisade and mesophyll cells both large and small chloroplasts are bright green in colour and distributed evenly throughout the cell. In leaflets showing orange frond the chloroplasts are golden yellow, but the arrangement of plastids shows little change. With orange spotting the chloroplasts are brownish-orange in colour and occur in aggregates, so that one part of the protoplasm may show a dense brown mass of plastids while other regions of the same cell may be completely devoid of them.

The chloroplast pigments of orange frond and orange spotting differ in their solubility in hot ethyl alcohol. When leaflets suffering from orange frond disease are extracted with boiling 95 per cent. alcohol the yellow pig-

ment rapidly dissolves, but in leaflets affected by orange spotting the pigments within the lesions do not dissolve.

From 1940 onwards the former Oil Palm Research Station carried out fertilizer trials on an area of palms with orange spotting in the Eastern Region of Nigeria. In one experiment dressings of 90 lbs. of wood ash per palm produced rapid and striking recovery shown to be due to the potassium constituent of wood ash by spectographic analyses carried out at the Rothamsted Experimental Station in 1945-46 and by recent chemical tissue tests. There is also an increase in the magnesium and calcium status of the leaves as the potassium content falls and the disease becomes more severe. These inter-relationships between different nutrients are now being studied in considerable detail, and it appears likely that the information so gained will be of great importance in formulating fertilizer programmes for wild and plantation palms growing in various regions of West Africa.

Before leaving the deficiency diseases the relationship between histological changes and symptom-reversibility may be briefly discussed. When magnesium is injected into leaves suffering from orange frond there is a fairly rapid suppression of symptoms but injection of potassium into leaves showing orange spotting fails to produce any change. It appears that in potasssium-deficient leaves the cells in the orange spot lesions are degenerate and the cytoplasm has collapsed. This is apparently an irreversible change and supplementary supplies of potassium cannot bring about the recovery of affected cells.

In orange frond disease, however, the cytoplasm appears to be intact but the chloroplasts do not contain the normal green pigments, but the latter can be synthesized in deficient cells when the magnesium deficiency is corrected. This explains the different responses seen in fertilizer trials with potassium or magnesium deficient palms. It is not possible to suppress the orange spotting on the leaves by the addition of potassium but the symptoms disappear gradually as new leaves are produced. In contrast to this, symptoms of orange frond disease can be cured dramatically by leaf or soil applications of magnesium sulphate.

Vascular wilt disease. We may now turn our attention to the parasitic diseases of mature oil palms, and we will first consider vascular wilt. This was first described by C. W. Wardlaw in 1945, and at that time it was feared that it might cause widespread losses in African plantations. It is fortunate that these fears have not so far been confirmed, and although the disease must still be regarded as a serious potential menace to the oil palm industry it is at the moment largely restricted and has not yet been recorded on wild palms in Nigeria. In plantations diseased palms may occur singly or in small or large groups, and obvious symptoms usually appear in palms from eight to fifteen years of age. Fronds which were previously erect and green suddenly wilt, bend downwards, become flaccid, and the rachis often breaks near the base, or at some distance above the base (sub-median fracture), so that the wilted leaves form a cloak around the upper part of the trunk. The speed with which wilt develops may vary greatly, and differences between "rapid" and "ordinary" vascular wilt presumably arise as a result of (a) variations in the susceptibility of various trees, or (b) variations in the pathogenicity of different strains of the fungus involved.

The causal organism is a strain of Fusarium oxysporum, which can be consistently isolated from diseased trees. The fungus ascends by way of the wood vessels, and in a longitudinal section of an affected palm brownish-grey

or black vascular strands can be observed, in marked contrast to the pale yellow or whitish strands found in a healthy palm. Necrotic brown or red streaks can be found in the vascular strands near the point where the roots join the trunk, and infection almost certainly occurs via the roots. In almost all palms suffering from vascular wilt the continuity of the necrosed vascular strands in affected roots and trunk can be demonstrated.

Inoculation experiments with the wilt Fusarium have been carried out exclusively on oil palm seedlings – which can be readily infected and show typical necrosis of the vascular strands, from which the fungus can be re-isolated. Infected seedlings, however, rarely show external wilt symptoms. They appear to tolerate the Fusarium; that is to say, they are 'infected' but not 'diseased'. Filtrates from Fusarium cultures, which might be expected to contain wilt-inducing toxins, also fail to produce disease symptoms in detached leaves of seedlings. This suggests that seedling palms are more resistant to the disease than mature palms, and inoculation experiments with the Fusarium will have to be continued for long periods so that any cumulative effects of infection can be discovered. Since the most promising control method for the disease lies in the selection and breeding of resistant types of palm these considerations may hamper the testing of progenies. Furthermore, results of pathogenicity tests on seedlings will need to be interpreted rather carefully, and arguing by analogy from seedling to mature palm will not necessarily be valid. Any programme of breeding for disease resistance must take account of inoculation experiments done on mature palms. With this end in view we are hoping to devise techniques for inoculating fully-grown trees with the pathogen.

Ganoderma trunk rot. A basal trunk rot caused by one or more species of Ganoderma is of widespread occurrence throughout the natural palm groves in Nigeria, but it is normally restricted to palms of more than twenty years of age. It has also caused considerable losses amongst old plantation palms in the Cameroons, and in parts of Belgian Congo. The first sign of the onset of Ganoderma trunk rot is a sudden and complete wilting of one or more of the lowermost fronds in the crown. As soon as wilting occurs these old leaves become flaccid and hang down, forming a cloak around the upper part and the trunk. In the later stages of the disease when perhaps half of the leaves have been killed the base of the trunk shows external blackening, gum may be exuded, and Ganoderma fruiting bodies begin to appear at the base of the palm. Sooner or later the rotting processes so weaken the trunk that the palm falls. The entire trunk is then rapidly destroyed and becomes covered with Ganoderma sporophores. The roots of affected palms are usually severely diseased. The cortex is brown and degenerate while the stelar tissues are jet black and presumably functionless. All the evidence so far available suggests that infection occurs primarily through the roots, but this has not yet been confirmed experimentally. At the Institute's Nigerian Sub-Station the heights of all the palms within one part of a wild grove were measured, and the presence or absence of Ganoderma infection was noted. Using height as an index of age it was shown that Ganoderma disease only becomes prominent after the palms have reached an age of about 25 years. Once a palm is older than 40 years it is passing into senescence, where death from Ganoderma or other causes is becoming increasingly imminent. In commercial practice the replanting of old plantations may give a large measure of control, but no direct method of controlling the disease in the wild groves has yet been devised. The problem is further complicated by the confused taxonomy of the genus Ganoderma making the separation of species difficult. Ganoderma lucidum is probably the chief causal agent in the oil palm belt proper but in the Cameroons the

disease appears on rather younger trees, and there the pathogen is often G. applanatum. It will be necessary to perform separate pathogenicity tests with isolations from different parts of Nigeria and the Cameroons, and a good deal of work remains to be done on the more detailed aspects of the Ganoderma problem.

Seedling diseases. Blast. The importance of raising healthy seedlings for establishing oil palm plantations is now generally recognised, and a study of seedling diseases forms an important part of the work of the Pathology Division of the W.A.I.F.O.R. The disease which has been studied in the greatest detail is one to which the name 'blast' has been given because seedlings affected by it appear to have been scorched by fire. It is present throughout the palm growing regions of West Africa, and at the W.A.I.F.O.R. Main Station causes important losses of seedlings every year in the months of November and December; they usually die within a fortnight of the symptoms first appearing.

In the earliest stage of the disease the leaves of affected plants lose their glossy appearance and become flaccid. The colour of the leaf then changes from dark green to an olive-green or khaki colour, and later to a clear yellow. Umber or purple colours then appear at the tip of the yellow leaves, and the discoloration spreads towards the base. After a day or two the whole leaf dies and becomes brown and brittle. When lifted from the soil the cortex of the primary roots is completely disintegrated, leaving the vascular strand hanging loose within the hollow cylinder of the hypodermis.

Although research on blast has been in progress since 1944 the cause of the disease is still unknown. At the W.A.I.F.O.R. an intensive search for pathogenic organisms began during 1951, and a most bewildering array of fungi was found. Fusarium oxysporum and other Fusarium spp., Chlamydosporium sp., Penicillium wortmanni, and Helminthosporium sp. were isolated but no one fungus appeared with any regularity and attempts to reproduce the disease in inoculation experiments gave very inconsistent results.

In 1951 the Cameroons Development Corporation sent a sample of roots from "blasted" seedlings to Rothamsted Experimental Station where a new species of the nematode Hoplolaimus was found associated with lateral roots but not in sufficient numbers to account for the extensive root damage found. During 1952 large numbers of the eelworm Aphelenchus avenae were observed within the rotten tissues. In 1953, further studies at W.A.I.F.O.R. Main Station gave no definite results. Both fungi and eelworms are present in the diseased roots, but eelworm populations show tremendous variation. The nematodes which occur most frequently are particulate feeders, while Aphelenchus avenae feeds on fungal hyphae; the eelworms extracted from "blasted" roots are, therefore, almost all sprobic forms and not obligate plant parasites. There is no indication of any correlation between root eelworm population and the occurrence of blast, or between soil and root eelworms populations in the rhizosphere of a "blasted" seedling. Attempts to reproduce the disease by the inoculation of eelworms into soil surrounding healthy plants have not given any consistent results. In field experiments soil fumigation with D.D. and parathion has been tested, and is of no value.

The present indications, are, therefore, that blast is not caused by eelworms, and further studies of the fungi associated with blast are now in progress. Curvularia and Nigrospora have been found on "blasted" roots,

and also a suspected Rhizoctonia species. The most promising discovery, however, has been a suspected Pythium species in the cortex of infected roots, but the fungus has not yet been isolated nor used in inoculation experiments. Pythium has not previously been recorded from the oil palm but may well be involved in the devastating root decay that is typical of blast disease.

Freckle. Finally mention must be made of two nursery leaf diseases. We will first consider that called freckle. This is of very widespread occurrence throughout Nigeria and the Cameroons, and it has also been reported causing severe damage to young palms in nurseries in the Congo, the Ivory Coast, and Dahomey. Freckle can assume epidemic porportions, and large numbers of seedlings are then destroyed. It can be carried over into the field when infected seedlings are transplanted and although it rarely kills young transplanted palms it produces general debility, and growth may be checked for some months after transplanting.

The first sign of infection is the appearance of groups of small, dark brown necrotic spots scattered over the surface of the leaflet. This is the non-aggressive phase of the disease, which is usually found on the younger leaves of affected seedlings. On the older leaves infection typically becomes aggressive. Tissues surrounding the primary (non-aggressive) lesions become dark brown in colour, and begin to necrose. The necrosis spreads rapidly throughout the leaf, which becomes pale greyish-brown in colour and ultimately dies.

French workers have claimed that freckle is caused by the fungus Cercospora elaeidis, and research done at the W. A I. F. O. R. Main Station has shown that Cercospora conidiophores are invariably associated with the non-aggressive lesions described above. A species of Cercospora has been isolated from diseased leaves and preliminary pathogenicity tests have indicated that the fungus is actively parasitic on seedling leaves. The life-history of the fungus and possible methods of control are now being investigated.

Anthracnose. The other leaf disease which is receiving attention is anthracnose. This occasionally causes severe losses and is probably responsible for many of the vacancies which appear after transplanting, and which in the past have been atrtibuted to "transplanting shock".

Dark brown blotches appear on the leaf and as these expand they become surrounded by an olive green or brownish halo. The centre of the lesion eventually becomes dry and bleached and assumes a pale greyish-brown colour. Minute black bodies (suspected acervuli) appear within the desiccated tissues, and the latter eventually die and shatter so that large areas of the leaf are destroyed.

The disease is almost certainly of fungal origin, and a fungus resembling Gloeosporium or Colletotrichum has been consistently isolated from diseased plants. The organism has not yet been identified but the acervuli, which are initially sub-epidermal, are furnished with setae and contain unicellular hyaline spores. There is a possibility that two strains or species may be involved, which differ in spore size. When growing on an artificial medium greenish black mycelium is produced and typical anthracnose lesions have been obtained by inoculating seedling leaves with spore suspensions.

Paper: Oil palm diseases in the Belgian Congo

by W.G. KOVACHICH
(Huileries Congo Belge)

Mr. Kovachich said that diseases in the Congo were similar to those in Nigeria, an exception being little leaf bud rot which was very prevalent in the former, but had not been mentioned by Mr. Allen. Wilt was the next most important disease and there was another known as "plant failure", which was thought to be due to malnutrition.

Ganoderma lucidum was common as also was a strain of Fusarium oxysporum, which attacked the folded young leaves. Armillaria mellea had been serious up to 1948, but had since then not been recorded in his company's plantations although it did occur in the nurseries. Two leaf spotting diseases also were known, due to Cercospora elaeidis and Helminthosporium halodes.

Little leaf bud rot caused malformation of leaves, arrested growth of the roots, and a complete rotting of the central stele of the palm. After extensive fertiliser trials it was tentatively attributed to boron deficiency.

Wilt disease was sometimes very severe, giving marked visual symptoms, which contrasted with the position in West Africa as described by Mr. Allen, and normally 90 per cent. kill could be expected within three months after dipping roots of young plants in a spore suspension of F. oxysporum. This was not achieved, however, by the inoculation of adult stems, where wilt symptoms were produced without the death of the plants.

Cortical root rot was prevalent in wet years in the nurseries, and he had found the sclerotia of Rhizoctonia constantly associated with the disease. He had not, however, been able to prove its pathogenicity.

C. elaeidis had been proved to cause Cercospora leaf spot, penetration occurring chiefly through the under leaf surface, the protection of which appeared to be necessary in order to obtain control.

Discussion: DR. KEYWORTH (Vegetable Research Station, Warwick) said that different effects were obtained by inoculating wilt pathogens into stems and roots and suggested that greater success might be obtained by inoculating the latter.

MR. KOVACHICH replied that it was almost impossible to inoculate the roots of adult oil palms.

MR. ALLEN confirmed that little leaf disease was not considered to be of economic importance in Nigeria.

DR. SNYDER (University of California) asked if anything were known of the host range of the oil palm Fusarium; would it for example infect banana?

Mr. KOVACHICH replied that he had not this information but that a collection of isolates from oil palm roots had been sent to Trinidad to the Banana Research Scheme for inoculation tests.

Paper: Blister blight disease of tea

by ERNEST HAINSWORTH
(Plant Protection Ltd., Fernhurst)

The first record of blister blight came from Assam in 1868 when S. E. Peal, who was a tea planter and a noted naturalist wrote in the 'Tea Planter' describing the disease and stated that it had been present in the Sibsagar district for some 10 years.

Towards the end of the last century specimens were sent by Sir George Watt and Dr. Harold H. Mann to Massee at Kew, who[1] named the causative fungus Exobasidium vexans.

The first severe epidemic of the disease was reported from Assam in 1895, and the disease again became epidemic in 1900. In 1908 it spread to the Darjeeling district, rapidly becoming epidemic throughout the area, and we read in the reports of that period that the future prospects of tea plantations and the possibility of replacing tea with some other crop were under active discussion.

However, the epidemic abated, and although the disease appeared every year subsequently on the youngest shoots, its importance declined. From 1890 up till 1947 it had almost disappeared from the plains of Assam, although in some tea-growing districts there were occasional reports of its activity. However, in September 1946 blister blight suddenly appeared for the first time in South India, and in the words of Dr. James Lamb[2], the Director of the Tea Research Institute of Ceylon: "It swept like wildfire through south India and the spores of the fungus, which are liberated in countless millions were doubtless carried by the prevailing winds to Ceylon". By October 1946 specimens of tea leaves attacked by blister blight in Ceylon had been sent to the Tea Research Institute, and by January 1947 the disease had spread over every tea area in Ceylon.

In April 1949 blister blight was reported[3] to have appeared in Sumatra, and although strict quarantine measures had already been laid down in Malaya it appeared there in February 1950[4]. In May 1951 it appeared in Java[5], and a year later it spread throughout that island to every tea estate so that by the middle of 1952 the disease was present in every tea-growing area in the East. In the northernmost tea-growing areas of Assam and Darjeeling the disease had never been of tremendous importance by reason of its seasonal nature, but as the disease came south towards the Equator and beyond it established itself on tea which was in production the whole year round, and on which there was always young growth susceptible to infection.

It is easy to see, therefore, the concern with which its appearance was greeted. At that time there was much speculation as to how the disease could possibly have been transmitted from north-east India to south India across 700 miles of hot plains, and against the prevailing winds. At first it was thought to have been brought via tea seeds, since south India and Ceylon have always brought in their tea from north-east India, and we got a request in 1948 that all tea seed exported to the south should be dressed with a protective seed dressing. It did not seem very likely that seed could be seriously considered as a carrying agent of the disease, especially as it had been sent for almost 100 years without trouble. Furthermore the system of certification of tea seed for export developed by A. C. 'Tunstall, which had

been in operation at Tocklai for 30 years, had failed during this period of mycological examination to reveal the presence of seed-borne Exobasidium. In any case basidiospores were not so long-lived as to be able to survive the journey of upwards 14 days from north to south. Other possible explanations of its transference to the south are linked with the establishment of air flight schedules between north and south India. Another interesting suggestion was that during the war south India loaned large amounts of estate labour to the Indian Tea Association for the construction of strategic military roads from Assam to Burma. When these men returned to their homes in 1946 many of them, being keen gardeners, took back with them plants from the new country in which they had been working, and it may have been possible that blister blight was transported on a young tea plant.

Germinating spores of Exobasidium penetrate the cuticle directly, the young leaves and stems being infected in this way. At maturity the epidermis is ruptured with the out-growth of the basidia to produce the characteristic white blister. Discharge of basidiospores is continuous for three to seven days and there is continuous and rapid infection of the blister by spores of Colletotrichum camelliae, the cause of brown blight, which produces blackening and eventual death. For many years conidia of Colletotrichum had been regarded as a conidia of Exobasidium, but the classic inoculation experiments of Gadd and Loos at the Tea Research Institute of Ceylon in 1949[6, 7], established that there were two fungi present in blackened blisters.

Coincident with the spread of blister blight through the southern areas the disease re-appeared and became much more widespread in the Assam Valley of north-east India. Up to 1947 it was regarded there as a curiosity, but now appears for a short time on very many estates each year. Control measures in north-east India are mainly agricultural and are aimed at the modification of husbandry practices so that there will be the minimum of young leaf on the plant at times of the year when the disease is normally present[11]. The general practice in estates where late season outbreaks were becoming a nuisance is now to postpone the commencement of the annual prune until the end of November.

Another interesting link between estate practice and the development of blister blight occurred as the result of the late arrival of manures. It was found that tea bushes receiving a heavy dose of manure in July or August continued to produce vigorous and succulent leaf until December, whereas bushes receiving their manure earlier, although not growing more slowly, were producing smaller and less vigorous leaves at that time. A combination of early manuring and late pruning was found to reduce outbreaks before April to negligible proportions. The practice of removing blistered leaves or leaves with incipient blisters in April and placing them on the ground has also reduced the amount of infection. With the establishment of these routines it has been found possible to reduce the amount of essential spraying to not more than four rounds of copper fungicide applied at seven to ten day intervals. Spraying is mainly confined to bushes which have been hard pruned and which consequently have vigorously growing shoots, and to nurseries at the time when the young stems first emerge from the ground.

In the rest of the tea-growing world the control of blister blight proved by no means so easy as in north-east India. Not only has active research into the disease itself been necessary but new spraying techniques have been worked out, wholesale changes in crop husbandry have been made, and vigorous efforts have been necessary to ensure that control measures were known and properly applied. Only last Friday, Mr. Wilson Mayne, Scientific

Officer of the Kanan Devan Hills Produce Company - large tea producers in south India - likened the coming of blister blight to South India in 1946 to the advent of coffee rust, Hemileia vastatrix, on coffee in Ceylon at the end of the last century.

First amongst the new measures has been the resurgence of interest in the tea plant as a horticultural producing unit. The old tea literature was searched for information about the behaviour of the plant in response to manures, and the effect of shade trees, mainly Grevillea robusta, under which tea was grown. The immediate plans involved the wholesale thinning and lightening of the shade and the adaptation for the first time of standard times for pruning and manuring so that more light and air would reach the tea plants, ensuring a minimum of new susceptible growth when conditions were most favourable for infection. These measures in their turn brought secondary problems. With the drastic reduction in shade came the appearance of red spider. With the exposure of rich soil carefully built up in the years of heavy shade came immediate increases in yields and the necessity for stricter observations of soil cultivation measures.

Meanwhile chemical control of the disease was being intensively studied and to augment the work of the research stations technicians from commercial fungicide manufacturers and crop machinery firms, both from this country and elsewhere, went out to work on various aspects of the problem. The campaign was vigorous and by the end of 1950 spraying or dusting tea for the control of blister blight had become routine throughout the affected areas. After four years' experience of chemical control measures the position now is that portable manhandled sprayers are used in close rounds of not longer than seven to ten days, right through the period of susceptibility. Some 16 to 18 spray rounds are being applied, each costing from 3s. to 5s. per round per acre, including the cost of labour. Dusting with power or hand dusters costs from 5s. to 7s. 6d. per round in the same numbers of round[11, 13]. The cost of fungicide represents over 90 per cent. of application costs when dusts are applied, but with spraying the cost of fungicide is only 25 per cent.[13].

Copper fungicides have been found after many trials to be the most suitable for blister blight control and copper preparations are preferred to the organic fungicides; mercury, of course, cannot be used on foliage which is to be consumed.

In general copper preparations are less expensive than organics and give higher yields and a higher degree of control. Satisfactory control can be achieved with copper fungicides applied by low volume techniques, using spray fluids containing 0·125 per cent. copper applied at rates of not more than 15 gals. per acre. Failure to apply control measures results in the annual yield declining by 50 per cent.

Now that control measures have become a routine there is interest in ways and means of cutting the fantastic cost of chemical protection. It costs over £5 per acre per annum to obtain adequate commercial control of blister blight. By closer attention to relative humidity and its relation to spore infection it should be possible to lengthen spraying or dusting rounds from time to time during the susceptible period. The use of power sprays and dusters is under active investigation[15]. Furthermore, we still have not reached the lowest limit of performance for some of our copper fungicides and we do not yet know how little copper we can apply per acre to achieve a reasonable degree of control, but blister blight is not now the nightmare which it was seven years ago, and plant pathologists can afford to feel proud

of the small band of their representatives in South India, Ceylon, Java, Sumatra, and Malaya who did not fail the tea industry in the anxious years.

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Paper: Blister blight of tea in Malaya

by ANTHONY JOHNSTON
(Department of Agriculture, Federation of Malaya)

Malaya was one of the last territories to be affected by the spread of blister blight which took place from 1946 onwards. The disease was first discovered in the highland tea-growing area of Malaya (Cameroon Highlands) at the end of February, 1950. There was a hope, although a very slender one,

that it might be possible to eradicate the disease before it had time to spread. An attempt was immediately made to do this but a survey of tea fields surrounding the original infection showed that already, by the time the disease had been noticed, occasional blisters could be found some distance from this point. It soon became obvious that immediate eradication was impossible and during 1950 the disease spread to all the other highland estates.

Climate and the disease. Climate has a very great effect on the incidence of blister blight. In the Malayan highlands seasonal differences are not clear cut; there are two vaguely defined wet seasons, a short one about April to May and a more intense one about October to December, but their severity, length, and time are all variable. Nor are the dry seasons marked, and completely dry spells of any length are uncommon. Because of this lack of dry season it was at first feared that the climate would be ideally suited to the development of the disease, which flourishes under conditions of high rainfall and high humidity.

It has become apparent during the last few years, however, that the climate is not so favourable as had at first been feared. Heavy attacks do occur, especially during the longer wet season at the end of the year, but the disease is to some extent held in check by the frequent occurrence of sunny spells in the mornings. These may be only of short duration but they do tend to occur commonly even in the wettest weather and help greatly in preventing heavy infections from building up.

Investigations. Several lines of investigation have been developed.

Spraying and dusting. First of all the problem of controlling the disease with fungicides was attacked. Basing our work on results already obtained by the Tea Research Institute of Ceylon and other institutes we soon found that good control under our conditions could be obtained by spraying with copper fungicides. Numerous fungicides of other types have been tried out but none has so far proved superior to the copper fungicides, the copper oxide type being particularly useful. In my experience blister blight is, in fact, easy to control with fungicides compared with other diseases occurring under the same conditions, e.g., late blight (Phytophthora infestans) of potatoes and tomatoes and leaf blight (Septoria apii-graveolentis) of celery. These require more frequent applications of a more concentrated spray, in very much larger quantities, for control.

The main difficulty in controlling blister blight lies in the application of the fungicide, and so far only pressure-retaining knapsack sprayers have been used extensively in Malaya. Because of the hilly nature of the ground and the lack of access paths it is difficult to use any apparatus that cannot be fairly easily carried.

Recently we have had some success with copper dust applied with hand dusters, and this method of control may prove more economical than spraying, by reducing the man-power required.

Resistance. We are naturally interested in the long-term possibility of obtaining resistant planting material. A selection programme is under way, resistance to blister blight being one of the characters sought. Considerable variation in susceptibility to the disease has been observed in the field and although no very resistant or immune bushes have yet been found it is hoped that useful material will ultimately become available.

Effect on yield. Another point which has interested us is the effect of the disease on yield. Information on this point is being sought by comparing the yield from bushes sprayed with a fungicide, and therefore free from disease, with that from unsprayed bushes. To obtain useful data it is necessary to take yields for a whole plucking cycle, which under Malayan conditions is usually $3\frac{1}{2}$ to 4 years.

Two problems are involved (1) the effect on yield of attack during the period of recovery from pruning and (2) the effect of attack on bushes during plucking.

Most damage is done to the bushes while they are recovering from pruning, and it would be expected that the effect of a severe attack at that time (in which practically all the new shoots may be completely killed off) would result in a severe set-back to the bushes, with consequent loss in yield. We have not yet obtained sufficient information to give conclusive figures but results so far indicate that loss of crop due to this type of attack is not as great as might be expected. Under local weather conditions the bushes are able to recover from a bad attack with great rapidity and although there may be an initial heavy loss of crop, this loss is gradually made up during the rest of the cycle.

As regards the effect on yield of attack by the disease during plucking there is some loss of crop under normal conditions but results so far indicate a loss of only a few per cent. It seems unlikely that treatment with a fungicide to control the disease in these circumstances would be an economic proposition, except perhaps for a few rounds during the worst weather at the end of the year.

At present I regard this work of assessing the effect of blister blight on yield as our most important line of investigation for until we know how much increase in crop can be expected from control practices it is impossible to advise growers on how much money they can profitably expend on these measures.

Discussion: DR. VASUDEVA (India) considered that in many cases the cost of fungicide was too high due to transport charges, and if possible local material should be utilized.

MR. HAINSWORTH replying to a question regarding the use of mist blowers to overcome the difficulty of applying liquid sprays said that he understood they were not effective at a distance of more than 20 yards, but that similar types of dusting machines had proved more successful.

MR. JOHNSTON did not favour the use of complicated power machinery by unskilled labour because of the heavy cost of upkeep. He preferred hand operated machines.

DR. TUNSTALL said that he had found the knapsack type satisfactory.

MR. SAMUEL (United Kingdom) asked about deleterious effects of spray residues on tea fermentation.

MR. HAINSWORTH said that when sprays were first used there had been some fear that the plucked tea might be adversely affected, but with the copper concentrations now used the residue from weekly applications did not exceed one third of the amount tolerated by the health authorities.

Meeting: Thursday, 22nd July, 10.0 a.m.

Chair: MR. F.C. BAWDEN

Subject: Virus diseases

Paper: Stone fruit virus diseases in Canada

by M. F. WELSH
(Division of Botany and Plant Pathology, Canada)

The title assigned to this paper was "Stone fruit virus diseases in Canada". A discussion at the present time of the causal agents of stone fruit virus diseases would be analogous to a report on the flora of an ocean floor after a short sail over the ocean's surface.

Dr. Berkeley presented to the Fourth Conference in 1948 an excellent survey of the stone fruit virus diseases recognised in eastern and western Canada at that time.

The period preceding 1948 had been one of rapid progress in the organization of virus surveys and in the recognition of virus diseases. The years since 1948 have yielded relatively few new diseases and have altered the status of only several others. These years can probably be described best as a period of consolidation. They have been marked by rapid strides in the organization of research, and by necessary alteration and elaboration of many of our concepts.

The organization of research has been accomplished chiefly by the development in the United States and Canada of committees, whose basis has been regional rather than national, each committee bringing together the laboratories and research agencies concerned with similar stone fruit virus disease problems. Thus the St. Catharines laboratory is associated with workers in the North Eastern and North Central regions of the United States, and the Summerland laboratory with workers in the states of the Pacific slopes of the continent. Through these regional groups a continent-wide organization has been established. This organization enables workers to keep informed promptly of the advances made elsewhere in a field of research that discourages frequent publication. A concrete result of such co-operation has been the publication in 1952 of the Stone Fruit Virus Handbook. So closely have the investigations of the various agencies been integrated that it will be impossible in this presentation to discuss stone fruit virus investigations in Canada without inclusion of information that has been obtained in other parts of America.

Our gradually changing concepts of virus diseases in stone fruits have been influenced most strongly by discovery of the widespread distribution of latent viruses in our tree populations and of an apparently unlimited variation in the behaviour of causal agents whose manifestations must for practical purposes be classified under a limited number of disease names.

Discussion of the latent viruses of stone fruits must begin with an attempt to describe the action of ring spots. These have been recovered from peach, sweet and sour cherry, almond, apricot, and various species and varieties of plum, but they appear to be most common on cherry. It is agreed among the investigators of the Western region that between 90 and

100 per cent. of the sweet cherry trees growing in Western Canadian and United States districts are carrying ring spot viruses. Most of these, termed necrotic ring spots, in both sweet and sour cherry, produce shock symptoms following infection, and thereafter become latent. A few are recurrent, producing symptoms in most or all seasons. The degree of symptom severity varies greatly, both for the shock symptoms of the so-called necrotic ring spots, and for the symptoms of the recurrent ring spots. The mildest expression, other than complete symptom masking, is a light-green or line pattern in the leaf lamina. The extreme expressions are characterized by the names "tatter leaf" and "lace leaf" that have been given to them by some workers. We presume that there are very many ring spot viruses or virus strains, but systematic separation and identification have not been possible. Most of the ring spot viruses produce severe shock symptoms in peach, and thereafter become latent in this host.

At least two other viruses are common latents in sweet cherry. One of these is sour cherry yellows, which under favourable conditions will produce symptoms in sour cherry. The second is prune dwarf, which is latent in both sweet and sour cherry. It appears probable that there exist strains of both. There is in addition a latent virus in sweet and sour cherry that causes a stunting and rosetting of peach. This virus usually, but not invariably, accompanies sour cherry yellows in the cherry. So far it has never been found unassociated with prune dwarf virus, providing a sort of negative evidence that the two may be identical.

The almost universal presence of latents in stone fruit material had enabled them to travel as contaminants in many experiments with economic diseases, and this has caused considerable confusion, especially in host range studies.

Equally great confusion has been caused by the many variations in symptoms and host ranges of the various sources of our economic diseases. An example has been the difficulty involved in determining relationships of the diseases that arrest ripening of sweet cherry in western North America. The disease little cherry that appeared in eastern British Columbia in 1933 invariably spreads very rapidly through inoculated trees, and has spread with great rapidity from tree to tree in the orchards. The disease that appeared subsequently in Washington and Oregon, and that is now known as western-X little cherry, produces almost identical symptoms in sweet cherry, but spreads more slowly through infected trees, and has a much lower rate of orchard spread. The behaviour of the two diseases is distinct in other hosts tested, including peach, Prunus virginiana var. demissa, and sweet cherry propagated on Prunus mahaleb rootstocks. Therefore, although the symptoms on sweet cherry are similar, little cherry and western-X little cherry are considered distinct diseases.

Most of the diseases of stone fruits provide similar, and sometimes even more confusing examples of symptom variation. In British Columbia twisted leaf of sweet cherry in some source trees produces leaf symptoms only. Other source trees show the same very characteristic leaf symptoms, but suffer in addition a severe distortion of fruits. Lambert mottle disease of sweet cherry displays slightly differing symptoms in almost every source tree observed.

Selected individual source trees for each of these diseases have been indexed on a wide range of stone fruit species and varieties. Such indexing has disclosed not only differing host ranges for the various isolates, but

also widely differing contents of abundant contaminant viruses.

Thus at least two possible explanations are available for the differing behaviour of the isolates. One is the influence of latent viruses on the host-parasite relationships of the specific virus, in other words the formation of virus complexes. The second is the existence of many viruses or virus strains able to produce symptoms of the same nature in a given host, with the contaminant viruses playing no role.

Experience with such phenomena is impressing stone fruit virus workers with the urgent need for development of means for separation of viruses from mixed culture, and more intimate studies of the viruses in pure culture. At the St. Catharines laboratory this has been reflected in the recently published work of Willison and Weintraub on the culturing and study of stone fruit viruses in cucumber, investigations which are continuing, and can now be elaborated by the use of recently-acquired electron microscope equipment. At the Summerland laboratory the same problem is being attacked by the formation of a team of plant pathologists, an entomologist, and a biochemist, in the hope that by the utilization of all the means of virus transmission, of virus inactivation, and of selective isolation available to such a team, means may be found for the separation in pure culture of many of the causal agents of diseases under study.

Concurrent with these investigations both laboratories have been continuing observations for the appearance of new diseases. Both laboratories are associated with local schemes for the production of virus-free tree fruit nursery stock. Both laboratories have active indexing programmes for the indexing of virus-free stone fruit clones. The St. Catharines laboratory for several years has been able to supply Ontario growers and nurseries with adequate amounts of sour cherry budwood, and is establishing sources of sweet cherry propagating material. The Summerland laboratory is associated with horticulturists of the Experiment Station in a stone fruit foundation stock project comparable to the British nuclear stock scheme. Both laboratories have had the assistance of entomologists in a search for vectors of stone fruit viruses. At the Summerland laboratory the testing of sweet cherry varieties for resistance to the little cherry disease has met with a measure of success.

The work reported from the St. Catharines laboratory has been that of Willison, Wintraub, Berkeley, and Phillips. That from Summerland has been carried out by Lott, Wilks, Wilde, Tomlinson, and Welsh.

[Dr. Welsh then exhibited a number of coloured slides of virus symptoms in various hosts.]

Paper: Virus diseases of pip and stone fruit trees in New Zealand

by E. E. CHAMBERLAIN
[Department of Scientific & Industrial Research, New Zealand]

Investigation of fruit tree virus diseases was commenced in New Zealand after the last war by Mr. J. D. Atkinson, Director of the Fruit Research Station, and assisted by Mr. J. A. Hunter, Technical Officer of the Plant Diseases Division. Work on many of the diseases is still in the preliminary stage but we have been able to show that there is a large number of pip and stone fruit virus diseases in the country.

The following list of virus and suspected virus diseases of fruit trees is an oversimplification of the position in New Zealand and will have to be considerably modified as we secure further information but it should form a basis for discussion.

Virus Diseases of Fruit Trees in New Zealand

Main Host	Name of Disease
Apple	Apple Mosaic
"	Apple Green Crinkle
"	Apple Ring Spot
Pear	Pear Stony Pit
Apricot	Moorpark Mottle
"	Apricot Fruit Distortion (tentative name only)
Cherry	Cherry Ring Spot
"	Cherry Rasp Leaf
"	Cherry Tatter Leaf
"	Cherry Aucuba Mosaic (possibly a strain of plum mosaic)
Peach	Peach Calico
"	Peach Rosette
Plum	Plum Mosaic

Suspected Virus Diseases of Fruit Trees in New Zealand

Main Host	Tentative Name of Disease
Apple	Gravenstein Gnarling
"	Apple Fruit Cracking and Bark Roughening
"	Apple Bark Blotch
Cherry	Cherry Marbling
Peach	Peach Crinkle
"	Stark Blemish
Plum	Plum Pox

As much information concerning the common fruit tree viruses is readily available I intend to mention only the more interesting aspects of these diseases in New Zealand. I will deal with the apparently new viruses in somewhat greater detail and conclude by giving a brief account of what we are doing to combat fruit tree virus diseases.

Apple Mosaic symptoms consist of creamy white spots, blotches or vein banding on leaves. Chlorotic areas sometimes become necrotic.

It is of wide distribution and high incidence, having been found on 43 standard apple varieties, five cider, two crab apple, and seven rootstock varieties. In Jonathan, Red Jonathan, and Golden Delicious it has not been possible to locate a single tree free from infection, while in a number of other varieties difficulty is being experienced in finding virus-free trees.

Experiments have shown that apple mosaic virus is systemic in a tree although symptoms may be restricted to one or two branches. There is also evidence that the virus occurs in a number of strains and that a mild strain can protect a tree from a severe strain. Thus in attempting to prepare trees for yield trials we have had considerable difficulty in infecting young trees with a severe strain of apple mosaic in spite of the fact that scions used

as a source of inoculum developed severe symptoms. It now appears that mild infection in the apparently healthy scions protected them from severe infection.

Field observations suggest that when symptoms are severe, vigour of trees and yield of fruit are considerably reduced. Loss of vigour caused by apple mosaic has been observed in several varieties but is most pronounced in Jonathan trees, which often develop severe symptoms. In some blocks of this variety up to 25 per cent. of trees show appreciable stunting and reduction in crop. Owing to the difficulty experienced in securing transmission of apple mosaic to standard varieties (both Jonathan and Ballarat varieties have been used) we have been unable to demonstrate reduction of vigour or yield in planned yield trials. We have, however, been able to demonstrate that a healthy seedling crab apple shows considerable reduction in vigour when infected with a severe strain of apple mosaic. As Hunter's Crab fruits prolifically we are setting out these trees in a yield trial, and at the same time we intend to establish further trials with a healthy seedling of the standard apple type.

Apple Green Crinkle is characterised by crinkling, dimpling, distortion, and russetting of fruits, does not appear to be widely distributed in other countries but has been recorded from Canada, where it is known as false sting, and also occurs in Australia. It occurs throughout New Zealand on the varieties Granny Smith, Dunn's Favourite, Cox's Orange Pippin, Gravenstein, Lord Wolseley, Northern Spy, Statesman, and Sturmer Pippin but incidence is rarely high. Granny Smith is the most commonly infected variety.

The disease is transmitted by grafting, and an interesting feature is that it is sometimes confined to a single leader of a tree for an indefinite period.

Apple Ring Spot, as far as we can ascertain, is confined to New Zealand. Symptoms, which occur only on fruits, consist of patches of rough russet bordered by smooth dark brown areas. Russetted areas may be quite small or may cover more than half the surface of the fruit. The dark brown areas may form narrow borders round russetted areas or may be extensive and form separate patches. In some cases they occur as concentric rings or broken rings when they form a pattern resembling thumb marks. Cracks occasionally form within the russetted areas.

Symptoms are common only on the Granny Smith variety although odd fruits showing similar symptoms have been found in several other varieties. Apple ring spot occurs on Granny Smith throughout New Zealand but other than in the Auckland district incidence is usually low and symptoms mild. In a number of Auckland orchards incidence is comparatively high, over 25 per cent. of the trees showing infection. In severely affected trees over 50 per cent. of fruits may be unmarketable.

The disease is perpetuated on scions from infected trees but it is several seasons before an appreciable number of fruits on the originally healthy trees show infection, and these are in the vicinity of the infected scions.

Apple ring spot is known to have been present in the Dominion for 20 years.

Pear Stony Pit in New Zealand has been found on the varieties Beurré Bosc, Beurré Clairgeau, Clapp's Favourite, Harrington's Victoria, Josephine, Clapp's Triumph, P. Barry, Winter Cole, and Winter Nelis. It occurs through-

out the Dominion and although incidence is usually low a few small blocks of trees show 100 per cent. infection. When infection is severe most fruits from an infected tree are unmarketable.

Observations and experiments in New Zealand confirm the views of Kienholz in the United States of America that the variety William Bon Chrétien is resistant.

Moorpark Mottle disease of apricots has apparently not been recorded outside New Zealand. Even in New Zealand it has been found only on five trees in a block of 89 Moorpark apricot trees in a Central Otago orchard.

Symptoms consist of well defined light green areas of varying shape and size, most pronounced on the upper leaf surface. The light green areas are sometimes arranged so as to form an ill-defined oak-leaf pattern. Symptoms are general throughout the tree but have not been observed on very young leaves and are therefore not evident in the early part of the season. Fruits are smaller then normal and distorted, having two depressions near the stem end on the side opposite the suture. They mature about a week later than fruits on healthy trees and are tough and leathery.

The virus has been transmitted by budding to seedling and Moorpark apricot, seedling and Paragon peach, and to cherry plum. On peach it causes severe shock symptoms in the year following infection, trees being stunted and leaves developing a reddening of margins followed by necrosis. Such leaves become tattered along the edges and fall prematurely. The young foliage also shows chlorosis. In subsequent years symptoms consist of a pronounced yellow vein banding of some leaves but growth of trees is almost normal. Fruits are small and late in maturing but do not show distortion.

Symptoms on cherry plum consist of light green or creamy white spots and line patterns on the leaves. Growth of trees does not appear to be affected.

In an attempt to eradicate the disease the five naturally infected trees and all our experimental material have been destroyed.

Apricot Fruit Distortion. This name has been tentatively given to a leaf mottling and fruit distorting virus disease which has been found on two Hemskirk apricot trees in a Central Otago orchard. Leaves on infected trees show light green to creamy white line patterns and are frequently distorted. On some leaves roughened reddish brown, corky tissue develops along veins on the upper surface. Fruits from infected trees show considerable crinkling and distortion.

The disease has been transmitted experimentally to seedling apricot, cherry, and peach and also to cherry plum.

On apricot seedlings in the year following infection the disease causes severe stunting, and rosetting and curling of leaves. In the second year after infection some shoots are killed while others are much stunted with small curled, necrotic leaves, some of which develop creamy white markings. On seedling sweet cherry, leaves at first show patches of light green vein clearing but later develop light-green to creamy-white spots, ring spots, and line patterns. Symptoms on infected peach seedlings are similar to those caused by plum mosaic, i.e., light and dark green mottling, chlorosis of leaf tips and margins, and indistinct line patterns of the oak tree type. On cherry

plum symptoms were also similar to those caused by plum mosaic.

This does not appear to correspond with any virus disease of apricots described elsewhere.

Virus diseases of cherry are prevalent in New Zealand and many trees appear to carry more than one virus. The position is by no means clear but we have been able to identify cherry ring spot, cherry tatter leaf, and cherry rasp leaf. These three virus diseases have been transmitted to seedling sweet cherry, and on evidence available they appear to correspond with virus diseases of the same names occurring in North America.

A cherry disease which we have tentatively named cherry aucuba mosaic causes a conspicuous bright yellow spotting, and ring spotting on leaves has been found on the varieties Florence and Werder's Early in several Central Otago orchards. This disease has been transmitted by budding to seedling cherry and peach and to cherry plum. Symptoms on these hosts suggest that it may be caused by a strain of plum mosaic.

Other virus symptoms have been observed on cherries but we have not yet been able to determine whether they are caused by distinct viruses, combinations of viruses, or whether they are merely different symptom expressions of the viruses mentioned above.

Peach Calico is similar to the disease carrying the same name in North America and is fairly common on peaches throughout New Zealand. Incidence is rarely high and in many cases symptoms are confined to a single branch or leader of a tree. The same disease has occasionally been found on nectarine.

Symptoms consist of creamy white to pale yellow blotches on leaves and in severe cases the greater portion of the foliage is chlorotic. Mild infection does not appear to affect vigour but when a large proportion of the foliage is affected the trees are stunted, young stem growth is chlorotic and pale areas are even evident on fruits.

Occasionally symptoms develop on branches adjacent to an infected scion in the year following budding but usually there is no transmission until the second year. Even then only some branches show symptoms.

Attempts to transmit peach calico to apricot and cherry plum have so far been unsuccessful.

In transmission experiments to sweet cherry seedlings one tree out of three developed symptoms of cherry marbling (a condition which will be mentioned later) in the second year.

Peach Rosette occurs in a number of Central Otago orchards on the varieties J. H. Hale and Muir. It is characterized by shortening of internodes and bunching of leaves, which are narrower than normal. Symptoms are most pronounced at the top of trees, new growth coming away at the base often being normal.

Rosette has been reproduced on infected scions and transmitted to healthy peach seedlings. Symptoms are most pronounced in the first or second years after budding and tend to disappear in the third year. The virus nature of peach rosette in New Zealand requires confirmation.

Plum Mosaic is the most widely spread stone fruit virus disease in New Zealand. It occurs throughout the country and incidence is high in many plum varieties. Plum mosaic is most common on plums but also occurs on apricots, peaches, almond, and probably on cherry. Incidence is so high on the variety Santa Rosa that difficulty has been experienced in locating virus-free trees. Plum mosaic as it occurs in New Zealand appears to be identical with the line pattern viruses of Spiro plum described by Willison in 1942 from Ontario.

Symptoms are extremely variable depending on species or variety of fruit tree, climatic conditions, and probably on strain of virus, but they usually consist of some form of chlorotic vein-banding, line pattern, or spotting.

Observations suggest that plum mosaic has an adverse effect on vigour of certain plum varieties. In nurseries infected cherry plum stocks are almost invariably smaller than healthy stocks while infected two-year-old Doris plum trees are noticeably smaller than healthy Doris trees of the same age. One Hawkes Bay grower stated that in a block of mature Doris trees, in which approximately 30 per cent. of the trees were infected, the healthy trees outyielded the diseased trees by five to one. An inspection of this block showed that the infected trees lacked vigour. A replicated yield trial using healthy and infected Doris plum trees has been laid down at Auckland, and these trees should start bearing next season.

An interesting feature of plum mosaic in New Zealand is the incompatability it causes in certain varieties. Thus Moorpark and Roxburgh Red apricot buds are incompatible with infected cherry plum stocks and there is evidence that other apricot varieties are also incompatible. With apricots incompatibility is not absolute as a low percentage of scions sometimes develop. The Japanese plum varieties Burbank, Sultan, and Wilson's Early, however, appear to be quite incompatible with diseased cherry plum stocks. Infected cherry plum buds also fail to survive when inserted into healthy trees of the above three varieties. It has also been shown that Sultan is incompatible with diseased Doris plum stocks. The Japanese plum variety Duff's Early Jewel shows a degree of incompatibility with diseased cherry plum, the bud take being reduced by nearly 90 per cent.

In addition to the above diseases the virus nature of which has been demonstrated by transmission there are in New Zealand a number of virus-like diseases [brief descriptions of which were given] whose nature has not yet been determined.

Amongst the pip fruits there is gnarling of Gravenstein apples, a fruit and bark cracking of the apple variety Kidd's Orange Red and a purple bark blotch condition which has been observed on Winesap apple trees.

Stone fruits are attacked by four virus-like diseases:- Cherry marbling, peach crinkle, stark blemish, and plum pox. Transmission trials are in progress for most of these disorders.

Control of Fruit Tree Virus Diseases in New Zealand

Fortunately we do not appear to have any serious fruit tree virus diseases which spread rapidly, and except for a few potentially dangerous diseases of low incidence such as Moorpark mottle, apricot fruit distortion, and plum pox we have not, as a general procedure, recommended destruction

of infected trees. Instead our efforts have been directed towards ensuring that all young trees sent out to orchards are free from virus infection.

The problem of producing virus-free trees is simplified by the fact that practically all trees for distribution to commercial orchardists are raised in one nursery, The New Zealand Fruitgrowers' Federation Nursery, which works in close collaboration with the Plant Diseases Division and acts on any advice we are able to give them concerning disease control. They are prepared to make determined efforts to produce virus-free trees. The nursery raises all its own stocks and secures its budwood from special trees which have been selected by Budwood Committees.

Although we have not yet succeeded in eliminating virus from the Federation Nursery we have been able to bring about a marked reduction in the number of infected trees despatched to orchardists.

In 1944 approximately 30 per cent. of the cherry plum stocks used for propagating plums and apricots were infected with plum mosaic. By roguing infected stocks the amount of infection in the stock beds has gradually been reduced each season, and when last inspected in February of this year only one infected stock was found in 9,000 examined.

Apple stool beds have also been rogued for appled mosaic and most of the stock varieties are now free from infection.

Budwood trees have also been inspected for virus infection and where possible infected trees have been replaced by healthy ones. This has not been possible with some apple varieties because no virus-free trees have been available. In such cases trees showing mild symptoms have been selected.

Unfortunately the above procedure has not been applied to private nurseries, who supply most of the trees for domestic orchards, and we consider that it is desirable to introduce some system of certified stocks and budwood trees to meet their requirements.

Subject: Fruit viruses in Great Britain

by A. F. POSNETTE
(East Malling Horticultural Research Station)

DR. POSNETTE said that the known virus diseases of fruit in Britain were roughly the same as those just described with a few exceptions such as cherry mottle and apple ring spot. The culture of most of these fruits was world-wide in distribution and, evidently as a result of the interchange of plant material before virus diseases were recognized, so were the diseases.

He proposed to discuss mainly the latent viruses, which were causing most of the losses in Britain; the really severe viruses were not prevalent. Rusty mottle of cherry was present in many trees but detectable in few, yet growth of infected trees was 20 per cent. less than that of healthy. The overall loss to the cherry industry due to this retardation of growth was very considerable.

At East Malling in a search made for virus-free clonal material of cherries and plums very few healthy ones were found; in some of the main

varieties, none.

Pear latent viruses might react vigorously with quince, seedlings of which were therefore used as indicators; by mass indexing of apples virus was found present in about 20 per cent. of the trees, and had quite a severe stunting effect on many varieties. The continued propagation of these latent viruses could only be avoided by using tested material.

Dr. Chamberlain had referred to the resistance set up by a mild strain of apple mosaic to a severe strain, and this was true in England; and not only with apple mosaic. Ring spot strains from cherry and plum provided protection against one another. This suggested a way of analysing a composite disease by using indicators infected by mild ringspot, which would probably keep out severe ring spot contaminants. By extending this principle we should actually get only the symptoms of the virus we are really interested in appearing in our test plants.

Another possible line of separation was by heat-treatment, about which little was as yet known at East Malling for stone and fruit trees; apple mosaic, however, was easily inactivated and possibly also cherry ring spot. In strawberries heat treatment was useful in separating viruses but was complicated by the extreme sensitivity of the test plants to the common strawberry viruses. By heat treatment for different lengths of time, however, it seemed possible to eliminate the viruses one by one, and by suitable testing after each treatment to ascertain the residual viruses. Such separation could not be attained by serological or differential vector feeding methods. It should be noticed that the majority of the diseases in the field were caused by a mixture of viruses.

The object now was to clean up nursery material in all parts of the world so that when new varieties were exchanged a collection of new viruses would not be sent at the same time.

MR. BAWDEN (United Kingdom) enquired about chemical tests for these viruses and also whether in the opinion of the speakers clean nursery stocks would eliminate the viruses, or was natural spread in the field too rapid.

DR. WELSH said that they were trying to find a practical chemical means of diagnosing virus diseases following Lindner's methods [R.A.M., 31, p.420], and using as far as possible the same material. This method seemed to distinguish between peach trees with symptoms and those without, but there was no practical application. It was then tried for cherry. Trees grown in the shade gave negative results whereas those grown in the light gave positive results. He thought essentially the same thing was true of peach.

MR. BAWDEN asked if others agreed that these tests might work in the laboratory but were not of practical use.

MR. JOHNSTON suggested testing by means of paper chromatography.

DR. CHAMBERLAIN said they had no experience of these tests in New Zealand.

DR. POSNETTE said the chemical test would appear to be useful for detecting infection, but was doubtful of its application to the identification of viruses.

DR. CHAMBERLAIN stated that the spread of these viruses had been greatly reduced in New Zealand by cleaning up the once central nursery which supplied

nearly all the commercial growers. When the work first commenced over 30 per cent. of the plum nursery stock was infected by mosaic introduced by infected bud wood. By systematically reducing infection in stock beds they had virtually eradicated viruses of plums and apricots. Considerable headway had also been made with apple mosaic by roguing.

The inspection of budding trees was performed by Budding Committees in various districts and much progress had been made. There was still a long way to go but the feeling was that if the nursery beds could be cleaned the general virus position in the country would be greatly improved.

DR. WELSH commented that in Canada there were diseases which spread in the orchard and others which apparently did not. Natural orchard spread of twisted leaf was sufficient to minimize the benefit of healthy stock, but the spread was slow and on general principles the fewer the diseased trees there were to start with the better. He considered that the inspection of source material would do much to reduce the serious effect of these viruses on fruit cropping. The ring spot position was similar, but spread was slower. Vigour of trees could also be improved by the elimination of latent viruses. For these reasons he favoured the certification of nursery source material.

DR. MAGEE suggested that a resolution should be formulated embodying the points made by previous speakers and particularly that only tested material should be used for international distribution.

MR. BAWDEN said that his question was not meant to suggest there was no need to clean up nursery stock. He wanted to be convinced that such methods would ensure healthy orchards throughout the world. He suggested that the recommendations should be referred to the Resolutions Committee.

This was agreed to (see Resolutions 5 & 6).

Continuing, MR. BAWDEN asked DR. POSNETTE how he estimated the 20 per cent. more rapid growth in healthy trees of a variety which was 100 per cent. infected with latent viruses, and also he was not clear why viruses from pear trees killed quince if all pear trees were now on quince stock.

DR. POSNETTE explained that the 20 per cent. decrease in growth was caused by inoculating healthy material of other varieties with the virus from the 100 per cent. infected varieties. The effect of the virus on the latter was unknown. Regarding the pear-quince viruses he thought that pears were more tolerant than quince and that the quince roots were in fact supported by the pear foliage.

Quince seedlings reacted strongly to the virus.

MR. BAWDEN asked what happened when the virus was inoculated to quince trees, and if it were only the seedling that reacted strongly.

DR. POSNETTE replied that large quince trees had not been inoculated but the stunting effect of the virus persisted in young quince; it was not a shock effect. They were trying to ascertain whether external causes such as soil conditions were implicated.

In reply to a question whether the dwarfing effects in stocks were due to the presence of latent viruses DR. POSNETTE replied that both infected

and healthy Malling-9 stocks produced dwarfing. Regarding pear-quince combination there was only circumstantial evidence as yet that dwarfing was due to the virus, rather than to endemical effect as with Malling-9. There was no evidence that the latter was due to virus. Replying to a further question DR. POSNETTE said that the only evidence he had of predisposition of a tree to spray damage as a result of virus infection was in the case of apple mosaic.

DR. WELSH cited cases in which virus infected trees had developed no symptoms during two years official quarantine. He considered that the only safe policy in plant interchange was certification at the point of origin.

Paper: Brassica viruses and diseases

by L. BROADBENT
(Rothamsted Experimental Station, Harpenden)

DR. BROADBENT said: In 1949 and 1950 in this country there was a very serious outbreak of cauliflower mosaic in most of the areas of England and Wales and some hundreds of pounds worth of crops were destroyed. As a result farmers began to recognise something was wrong and we were asked if we could do anything to learn more about this disease. I would stress that this work has been essentially co-operative. We have attempted to study it in all the broccoli-growing areas of the country, and in addition we paid attention to cabbage black ring spot which is not so common in this country, for reasons which I will tell you later. I would just remind you of some of the symptoms. On most cauliflowers cabbage black ring spot does not give necrotic but chlorotic symptoms, though occasionally it goes necrotic. Only on the older leaves on some plants of some varieties is it necrotic.

Cauliflower mosaic starts by vein clearing, and later develops leaves showing vein banding. When both viruses are present together you get mottle. There are many types of mottle associated with both viruses and invariably, as I say, the plant is infected with both. And both viruses are present in a number of strains, from mild to severe. Cabbage black ring spot is usually overlooked unless it is present in a severe strain. Cauliflower mosaic can be masked, but in this country of low temperatures we have never seen it masked.

Plants like thrift are very good indicators of strains. The mild strain will only cause the usual vein-clearing whereas the severe strain of both viruses will kill. We have studied the symptoms in different crops by infecting them artificially, and this we found extremely valuable, not only for ourselves but also for the plant pathologists of the National Agricultural Advisory Service. One other symptom is that of stipple, which was brought to our attention by van Koot of Holland, where it causes a great deal of trouble in their cabbages over-wintering in cold store. It had not been recognised as a symptom of cauliflower mosaic for the simple reason that only a few varieties show it, and only one or two plants occasionally of those varieties.

Now with regard to the spreading of these viruses, let us consider first of all, spread into the crop, on which Dr. Storey did quite a lot of work in Yorkshire. We found that relative isolation (and I am not going to define relative) made a considerable difference. In areas like Wakefield, where the crop is grown in small holdings with other brassicas on the same farm and late varieties are grown so that the mature crop overlaps with the seed-bed,

you find ample opportunity for viruses to be spread from one crop to another, and it was in such areas that the worst outbreaks occurred. In Cornwall, on the other hand, where the broccoli-growing is somewhat isolated and other brassicas are not grown to any extent (and those that are grown are usually out of the ground before the seedlings come up) then cauliflower mosaic is almost absent. Weeds, we have found, are not important, possibly because so many of those that take infection, both of cabbage black ring spot and cauliflower mosaic, die.

Spread within the crop. Most of the infection is within about eight plants of the source of the infection although occasionally vectors move farther than that. The pattern of spread rises from half a per cent. to 41 per cent. during the season from late autumn to spring. Many plants become infected during the time of autumn migration of aphids but all fail to show symptoms until the spring but the loss from these late infections is negligible. The greatest loss in yield is from plants that become infected early, either in the seed-bed or soon after they are planted out. From that fact it is fairly obvious that one should try to protect the seed-bed as much as possible because you plant out infected plants which may not then be showing symptoms, and the more there are the greater the number of sources within the crop and therefore the quicker the subsequent spread.

To give an example of the first type of protection, namely, isolation, as used by Dr. Storey in Yorkshire. She had one seed-bed high in the Wolds and another one in a Wakefield holding where most of the growers, of course, raise their own seedlings. When these plants were later put out there was no infection whatsoever from the Wolds, whereas there was nearly six per cent. infection in the Wakefield crop. We have tried most of the present day insecticides, both contact and systemic, and not one has had any effect whatsoever. It is practically impossible to prevent the introduction of quickly spread viruses by insecticides. Until the chemists produce some repellants it is no use, I think, going further there.

Then we attempted to protect the seed-bed with barriers and found that crop barriers were best when they were narrow rows, say, two or three rows of cereal or other brassicas. Other brassicas are not so good, of course, because they become infected and the infection can be taken from them into the seed-bed. We found cereals the best barriers. Aphids carrying the virus would land on them. If they feed for any length of time, as they will do occasionally, they lose their power to transmit the viruses. Even if they do not feed they take off again, and when an aphid takes off from a plant it does not like it generally flies upwards and away so it will probably fly over the seed-bed. Seed-bed trials and the incidence of cauliflower mosaic trials were all done on small seed-beds. This year we have extended it on a commercial scale, but one or two farmers have been protecting their seed-beds in this way and have got extremely good results to date. In two experiments in Kent and Devon last year we had eight per cent. mosaic in the unprotected and two per cent. in the protected seed-bed. Moreover, there was a greater percentage of plantable plants in the protected seed-bed. They grew better and a higher proportion were planted. The results depend to some extent on the width of the barrier because if the seed-bed is too narrow, there is no air movement and the plants are drawn and poor. Finally we have investigated the spacing of the plants in the seed-beds, and we found the usual result one gets with viruses - the closer the plants are to each other in the row or between the rows the smaller the proportion of infected plants. In other words the same number of plants become infected but they are fewer in relation to the total.

Now with regard to susceptibility and tolerance, it is my opinion that the future control of these diseases lies in the development of tolerant or resistant plants. And here the National Institute of Agricultural Botany and the plant breeders are co-operating. For instance, we found with two cauliflower varieties, Continuity and Mid-Summer, that each originally had about two per cent. infection from the seed-bed. At the end of the season Continuity had 15 per cent. infection, whereas Mid-Summer had 35 per cent. And we found that on other occasions Continuity was not only more resistant but more tolerant. This applies to certain other varieties. Majestic suffers very badly indeed from cauliflower mosaic but All-the-Year-Round does not suffer to anything like the same extent, although it will be infected at the same time with the same strain of virus. There is more hope of obtaining tolerant varieties, of course, than resistant varieties, but resistance is present in some of them and it may be possible to work it up.

With regard to vectors we have found about a dozen new vectors, which raises the number described in the literature, I think, to something like 25. But we found with the field work that only when Myzus persicae and Brevicoryne brassicae are present in number are the viruses spread. The other vectors can cause occasional transmissions but they are of no great economic importance. M. persicae is just as important as B. brassicae as a vector, but it is rare in this country for it to be so numerous as brassicae. What I mean is that it never gets into the plague category that brassicae gets into (e.g., in 1949 and 1950). Why does cabbage black ring spot not spread so rapidly as cauliflower mosaic? So far as we know, though we know little about its concentration, it is present in the south in more highly concentrated conditions. It has a very much wider host range and, except in some years, it is relatively scarce. There are two reasons. Mrs. Watson and her colleagues have recently found that it is quite likely that cauliflower mosaic virus is retained by the aphid, and therefore once the aphid has picked it up it may be able to spread it to more plants than if it had just picked up cabbage black ring spot. More important still, in my opinion, is the relative distribution of the two viruses within the plants. Cauliflower mosaic is distributed throughout the plant, while cabbage black ring spot, once it has become established, is concentrated in the lower leaves of cauliflowers.

We have also tried roguing and applying effective fertilisers. The more nitrogen you give to plants the greater the amount of disease you will get, and the winter death is probably not, as has been assumed, associated so much with virus infection as with high nitrogenous state. Where you have a low nitrogenous status they winter just as effectively as the diseased ones; where the nitrogenous status is high healthy plants die just as diseased ones.

Recently we have been working on various yellow mosaics. First of all Mr. Ogilvie sent in to us a plant of honesty with broken coloured flowers, thinking it was cabbage black ring spot. It was not. It turned out to be a strain of turnip yellow mosaic, described by Kenneth Smith in Edinburgh. Then two years ago there was a plague of flea beetles in most of England and in that autumn a new disease turned up in the Newcastle area. This was first confused with cauliflower mosaic, but it was soon obvious that it was not that. Again we found out that it was a strain of the turnip yellow mosaic, but there was also something else present. Dr. Blencoe is doing work on these viruses and he considers there is something else present which is not the Cambridge virus, as we call it, that came from Edinburgh. This Newcastle virus infects cauliflowers, cabbages, kales, and brussels sprouts readily whereas the Cambridge one does not, therefore it is potentially much more serious. But

it is beetle-transmitted, as is the Cambridge one, and therefore should be relatively easy to control with DDT. Finally, we found what appears to be a new virus, a turnip yellows, in Edshill in Scotland, which is not related to the Cambridge one. It is present in high concentration. It is also transmitted in the field by contact. We do not yet know what to call it but we have not finished working on these viruses yet.

(Coloured lantern slides of these virus diseases were shown.)

Discussion: DR. CHAMBERLAIN said that cabbage black ring spot had been present in New Zealand for many years, but cauliflower mosaic had only recently been recognized. It appeared to be on the increase. At present it was troublesome on swedes and turnips and threatened to become destructive in the future on cauliflowers.

DR. BROADBENT said in answer to a question by DR. SILBERSCHMIDT that he had not had much experience with the viruses in Chinese cabbage, but the variety they used for work with turnip yellow mosaic suffered badly both from cauliflower mosaic and cabbage black ring spot, and so did turnips and swedes. The effect of cauliflower mosaic on kale varieties was variable.

DR. HOPKINS (C.M.I.) said his experience with these two virus diseases in Southern Rhodesia was almost identical with that of DR. BROADBENT. As long ago as 1943 he had published a note on his investigations, but virus nomenclature was so confused at that time that what to-day was called cauliflower mosaic was then known as the cabbage mosaic of Walker and Larsen. In consequence he had concluded his paper by saying that there was no evidence of cauliflower mosaic in Southern Rhodesia. That statement had now to be reversed. He had suspected the presence of cabbage black ring spot but was unable to confirm it owing to lack of facilities. He had had no difficulty in transmitting both viruses by Myzus persicae and Brevicoryne brassicae, which were very prevalent in seed-beds, and had only been able to raise healthy plants during hot weather when aphids were very active by spraying 14 times with nicotine sulphate before transplanting, which was impracticable. He had found Myzus persicae at the cotyledonary stage in seed-beds so that spraying had to commence before germination took place. He had concluded that in order to grow healthy cabbage successfully a periodical "closed season" of about four months was necessary during the hot spring weather, or 90-day varieties only should be grown at that time. He was not sure whether these varieties showed some resistance or whether they merely missed the virus by their early maturity. He had found low temperatures to mask cauliflower mosaic, and this allowed cauliflowers to be produced during the winter. During hot weather cauliflower mosaic virus was highly destructive to cauliflowers but less so to cabbages.

DR. STOREY asked if transmission of the viruses by flea beetle was purely mechanical.

DR. MATTHEWS said that in serial transfers flea beetles transmitted immediately, then did not do so for a 24 hour period, after which they continued to do so for 3 or 4 days.

MR. BAWDEN said the flea beetle should be more effective than the aphid because the former could remain infected for days, whereas the latter could not.

DR. BROADBENT said there was a very different rate of spread in young plants infected by these two viruses. Aphids could retain the cauli-

flower mosaic virus longer than that of cabbage black ring spot, which was an important point. He still maintained that the relative distribution of viruses in the plant did play a part.

DR. MARION WATSON (Rothamsted) (referring to flea beetle transmission of turnip yellows) said: The success of a vector depends not only on individual efficiency but on the proportion of insects which become infected and the numbers available. The same sort of reasoning applies to Dr. Broadbent's argument concerning the relative levels of transmission of cabbage ring spot and cauliflower mosaic viruses, but in reverse.

Cabbage ring spot (CBRS) virus is non-persistent and needs a large number of vectors coming from a large nearby source of infection to cause large spread of virus. Cauliflower mosaic (CLM) is transmitted in short feeding periods like a non-persistent virus, but there is evidence that it survives for much longer in its aphids (when feeding) than true non-persistent viruses and is therefore more of the nature of a persistent virus. If this is true it could be transmitted equally as efficiently as CBRS in conditions where CBRS is being transmitted, but could also be transmitted in conditions unfavourable to the transmission of non-persistent viruses, i. e. with fewer vectors and from a distant source. It is unlikely that the more restricted spread of CBRS depends on its localisation in the outer leaves of the plant because beet yellows virus is most efficiently transmitted by the same vector and is also mainly located in older and outer leaves of its host (sugar beet),

Paper: Wheat streak mosaic in Canada

by J. T. SLYKHUIS
(Lethbridge, Alberta, Canada)
[Presented by W. C. Broadfoot, Canada]

Wheat streak mosaic was recognized in Alberta, Canada, for the first time in 1952, and was particularly widespread and destructive in 1954 when it occurred in substantially all winter wheat crops in southern Alberta and caused moderate to total financial loss of approximately 25 per cent. of the winter wheat crops in areas where winter wheat has been grown generally for several years.

An eriophyid mite, Aceria tulipae Keifer, was proved to be an efficient vector of the virus. There is evidence that this mite carries an additional factor causing severe chlorosis, necrosis, and stunting. This factor differs from wheat streak mosaic by not being manually transmissible. The mites themselves cause a longitudinal rolling of wheat leaves, but when non-infective they do not cause chlorosis unless they become very numerous.

When reared on diseased wheat all stages of A. tulipae except the eggs carried the wheat streak mosaic virus. Mites remained infective for six days on an immune host. Although nymphs acquired the streak mosaic virus by feeding on manually-infected wheat plants adults, that could be distinguished from nymphs by their darker colour and larger size, did not.

Wind is a major factor in the dispersal of A. tulipae and hence of the wheat streak mosaic virus.

Although A. tulipae transmitted the wheat streak mosaic to a number of wild as well as cultivated annual grasses these mites multiplied readily only

on wheat. Sparce colonies were maintained for several weeks on Poa compressus and Oryzopsis hymenoides, both of which can be infected with the virus. A.tulipae was found in nature on the perennial grasses Hordeum jubatum, Agropyron smithii, and Elymus canadensis, but attempts to rear mites from these grasses on wheat were unsuccessful.

All stages of A. tulipae survived the winter on winter wheat. The mites survived lower temperatures than hardy varieties of winter wheat artificially hardened and exposed to various sub-freezing temperatures in temperature-controlled rooms.

Both the wheat streak mosaic virus and A. tulipae are substantially dependent on living wheat for their perpetuation through winter. During the spring and summer the virus can spread with the mites to spring wheat and other susceptible plants. Volunteer wheat growing on summer-fallow land or at the edges of fields may also perpetuate the virus and its vector during the summer and fall to endanger winter wheat sown in or near fields in which diseased plants are still growing. The disease can be effectively controlled by avoiding unnecessarily early seeding of winter wheat and by eliminating all living wheat plants in or near fields in which wheat is to be grown. Both the mites and the virus are destroyed on diseased plants killed by cultivation. However, the mites survive long enough on infested leaves buried in or placed on the surface of the soil to infest wheat seeded up to six days later. Since the mites soon perish on leaves that become desiccated or partially decomposed the rate at which they are eliminated on plants uprooted by cultivation is influenced by temperature and moisture conditions in the soil.

Discussion: DR. WELSH asked for information on spraying methods for the exclusion of mites.

DR. BROADFOOT replied that DR. SLYKHUIS could supply such information.

MR. BAWDEN asked if streak mosaic was the most important virus disease of wheat in Canada.

DR. BROADFOOT said that rosette disease was not present in Western Canada and no other virus disease of wheat was of economic importance there.

MR. BAWDEN asked if DR. SLYKHUIS thought there were three mite-transmitted virus diseases.

DR. BROADFOOT said he thought only two, the manually and non-manually transmitted.

DR. HANNA said the most damaging virus was transmitted by the mite, but could also be manually transmitted.

MR. MILLIKAN asked whether other cereals were affected and whether there was any known resistance.

DR. BROADFOOT replied that Triticum dicoccum, T. aestivum, and Elymus canadensis were known hosts, but that winter rye was practically resistant.

Meeting: Thursday, 22nd July, 2.30 p.m.

Chair: MR. F.C. BAWDEN

Subject: Virus diseases (contd.)

Paper: Tristeza and other virus diseases of Citrus in New Zealand

by E.E. CHAMBERLAIN
(Department of Scientific & Industrial Research, New Zealand)

The study of citrus virus diseases, which is in its initial stages in New Zealand, has been planned as a joint project. Field surveys are being carried out by Dr. H.M. Mount, of the Fruit Research Division, and Mr. W. Fletcher, Citriculturalist, Department of Agriculture, while Mount and I, with the technical assistence of Mr. J.A. Hunter, Plant Diseases Division, are responsible for the experimental work.

Citrus tristeza: This disease, although undoubtedly prevalent in New Zealand, has not become of economic importance as we do not use sour orange as a rootstock. Indeed we were not aware of its existence in the country until rootstock trials, which included sour orange as a rootstock and as an intermediate, were commenced by Woodhead and Mount in 1938. Various combinations of Washington Navel sweet orange as the scion with sour orange, trifoliate, rough lemon (citronelle), and Cleopatra mandarin as stocks or intermediates were used in these trials. In the first year after budding growth of the Washington Navel scions, which were clonal, was apparently normal in all combinations. In the second year, however, in all combinations in which sour orange was present as either rootstock or intermediate the scions failed to make further growth and gradually declined. Eventually most scions died.

Similar symptoms developed when late Valencia sweet orange and certain mandarin varieties were grown on sour orange rootstock. Some sweet orange varieties, however, including Carter Navel, were apparently free from infection and did not suffer decline but over a period of years many have developed typical tristeza symptoms - yellowing of leaves, setting of a very heavy crop, leaf fall, decline, and eventually death. This indicates that the disease spreads naturally in New Zealand, which is to be expected since the chief vector, Aphis citricidus, is prevalent and A. gossypii is also present.

The presence of tristeza in New Zealand has been confirmed by glasshouse trials commenced in 1952, using Washington Navel on sweet and sour orange stocks.

Last year trials were carried out to ascertain whether New Zealand grapefruit could be safely used as a rootstock for sweet orange when tristeza was present. Preliminary observations suggest that the disease may cause stunting of sweet orange on New Zealand grapefruit. This requires verification. It is to be noted that New Zealand grapefruit is not a true grapefruit but probably a chance hybrid, with the possibility of having mandarin, sour orange, and pummelo blood in its makeup.

Stem pitting is now thought to be an expression of tristeza. In New Zealand stem pitting symptoms have been observed on trees of Marsh grapefruit (stock either sweet orange or rough lemon) and on intermediate stem pieces

of Morton citrange (trifoliate × sweet orange). Symptoms were very severe in the record case which involved young trees 6 to 7 years old of Carter Navel on trifoliate stocks.

McClean, of South Africa, reported that New Zealand grapefruit trees sent to him gave symptoms of stem pitting when indexed on lime stocks. Trees from which budwood was taken were 20 years old, vigorous and showed no symptoms of stem pitting even when bark was stripped from the trunk and large branches.

Scaly butt. In U.S.A. this disease is also known as exocortis. Symptoms, which consist of a scaling of the bark below the bud union, occur only when trees are worked on trifoliated stock (Poncirus trifoliata). In New Zealand this disease has been observed on Temple, an American variety thought to be mandarin × sweet orange, on Murcio, on Australian mandarin, and on Persian lime. There appeared to be stunting of trees of Temple, those of Murcio were young and have since been removed; the Persian limes seemed to be fairly vigorous.

Bark blotch of lemons is very common in New Zealand. The variety Eureka is particularly susceptible and it is doubtful whether any tree of this variety over 18 years of age is free from infection. Symptoms consist of scaly patches on the bark. Isolations from affected bark usually yield Ascochyta corticalis and in the past this fungus has been considered to be the cause of the disease. It is probable that the disease is shell bark, which is generally considered to be a virus although this has not been definitely proved.

Psorosis (or Scaly Bark). Although a definite watch has been kept for bark symptoms of psorosis in New Zealand we have no definite evidence of its occurrence. As the disease is of almost universal distribution it is unlikely that it is absent. Psorosis produces foliage symptoms in the early spring and we intend to make a survey in an effort to discover whether the disease is present or not.

A bark condition of New Zealand grapefruit similar to some forms of psorosis is widespread, and the same condition has been reported by Dr. Lilian Fraser from New South Wales. However, affected trees of New Zealand grapefruit top-worked to navel orange show no bark symptoms on the new top, so it cannot be ordinary psorosis.

Recently a further virus-like symptom has been observed on citrus in New Zealand; a severe stem pitting resembling flat limb or gnarling of apple. This occurred on Surprise Navel (a sport from Washington Navel) trees on rough lemon stocks.

Paper: Lime die-back in the Gold Coast

by W.A. HUGHES
(Late of Department of Agriculture, Gold Coast)

The lime industry of the Gold Coast is concentrated in the fringing forest region behind Cape Coast. The West Indian lime was sown by the Department of Agriculture in 1913, and its cultivation adopted by the local farmers. The limes were grown from seed in small individual farms and to such an extent that export of raw lime juice became possible, indeed during the 1930's over-production of fruit occurred. There are few plantations in the rest of the

country, but individual limes are to be found in villages throughout most of the country.

The decline of the limes was first observed by 1938, and this unthriftiness increased until production fell and areas of trees started to die out.

Preliminary work indicated that a species of Trametes and Ganoderma lucidum, two bracket fungi which are common on moribund trees, were not responsible for the decline. Pot experiments dealing with micro-element deficiencies did not reproduce the symptoms of the disease, nor could any evidence be found for bacteria or fungi as causal agents.

It was not until Lister grafted tissue from diseased limes into healthy seedlings that the symptoms of the disease could be reproduced experimentally and it was concluded that a virus was responsible. Lister also initiated investigations on the alternate hosts and the anatomy of diseased trees.

The leaf symptom consists of a marked clearing of the veins. In the field the clearing is usually discontinuous along the veins, giving a "flecked" appearance. The first symptom on inoculated plants consists of a striking clearing of all the leaf veins, which in succeeding leaves fades into flecking; and eventually symptomless leaves may be produced. The symptoms in the field are variable, as infected trees may produce symptomless leaves at one season, but at other times distinct cleared lengths of vein may be seen in the leaves.

A corky enation on the upper side of the leaf along the veins has been observed but, although associated with the disease, it was not transmitted by grafting and is rare in the field. Infected trees tend to die back from the top, giving a crown die-back after defoliation. More common is a side die-back, giving a lop-sided appearance to the diseased tree. Sectional die-back is common. In this, a strip of dead bark can be traced from a dead root up the side of the trunk, along the underside of a branch to dead twigs at its distal end. The branch, although dead on the under side, bears living shoots above. This condition is probably associated with pitting of the wood, which is often seen when the bark is peeled from diseased trees. The pitting in the xylem is not associated with sunken areas in the bark.

In sections of diseased seedlings, Lister found that the gumming appears to start in the cambium which produces large thin-walled cells in wood and phloem; the contents of these appear to degenerate and form particles of gum. Further degeneration of these cells results in the formation of a mass of gum with isolated cells floating in it. Individual wood vessels become plugged with gum and a longitudinal section shows that several plugs may exist in the same vessel. Finally, a sector of the wood is completely broken down to give a wedge-shaped gum plug in the xylem, and if this condition is extended a strip of dead bark may be formed.

There is another form of the disease in which the plants are dwarfed and the size of the leaves are reduced, but die-back is not common. Lister obtained indication that this may be caused by a different strain of the virus, but it was not possible to investigate this fully.

Inoculation of the virus into other Rutaceae was performed by grafting seedlings of the species or variety and then several months after the graft had taken, grafting back to healthy lime seedlings as indicators. The work was done in the open, with ungrafted seedlings as controls. In this way rough

lemon, lemon, Trinidad wild grapefruit, and Clausena anisata were shown to be symptomless carriers of the virus. Sour orange became chlorotic and growth was retarded, but the infected plants lived for two years after being grafted, when they were discarded. Aegliopsis chevalieri showed distorted leaves and vein clearing, while two out of four showed corky vein, but this condition was not transmitted to lime indicator plants. Afraegle paniculata showed vein clearing but no leaf distortion and again some of the plants produced leaves with corky veins. Citropsis articulata was killed by the virus. The symptoms in these rutaceous plants took up to six months to develop although grafting was done when the plants were in active growth.

Sweet citrus on sour orange, suffering from a condition similar to tristeza, were grafted to healthy lime seedlings which later developed the leaf and die-back symptoms of the lime die-back; Washington Navel, Satsuma, Tangerine, and Marsh grape fruit were tested thus. The reverse graft was not possible as no healthy sweet citrus was available.

Aphis citricidus, which is very common on citrus in the Gold Coast, was shown to transmit the virus in six out of ten test plants, the symptoms appearing in one month. A mealy bug Ferrisia virgata also transmitted the virus. When taken from limes in the field and fed on source plants for 30 hours, six out of eight transmissions were obtained, with one control infected. When fed for 24 and 3 hours in succeeding batches and then placed on the test plants, nine out of ten transmissions occurred with no infected controls. A faulty insectary may account for the control infection. Aphis maidis did not transmit the virus, although it appeared to feed quite happily on the limes.

With regard to the control of the disease the presence of an ubiquitous and highly mobile vector, together with an almost universal infection of the crop, rendered roguing impossible. However, limes budded on rough lemon stock about 10 to 15 years previously were found to be still bearing a crop and it was decided to replace the moribund seedling limes with budded stock. The scion wood was taken from diseased seedlings but made good growth and produced a crop.

Finally, I should like to express our thanks to all who helped, the farmers, local firms, other members of the Department, and in particular the staff of the West African Cocoa Research Institute, Tafo, who so generously gave help and guidance, the responsibility for all statements being that of the speaker.

Paper: Virus die-back disease of limes and other citrus in the Gold Coast

by E. B. MARTYN
[Presented by J. M. Waterston, Nigeria]

SUMMARY

A die-back disease of West Indian limes was first recognised in the Gold Coast in 1926, probably introduced on citrus budwood from South Africa. In 1948 it was shown to be graft transmissible and is now considered akin to the virus diseases of citrus known as 'stem pitting', 'tristeza' and 'quick decline'. It is transmitted by Aphis citricidus, which occurs commonly, and the mealy bud vector of cacao swollen shoot, Ferrisia virgata, has been suggested as a possible other vector.

The disease, which causes a rapid die-back of limes on their own roots, resulted in the virtual extinction of a small lime industry in the Cape Coast area. It also kills sweet orange, Marsh grapefruit, and eventually lemons when these are on sour orange stock. Marsh grapefruit and lime on rough lemon stock become affected too, the latter starting to die back within five to six years. Severe pitting is occasionally but not universally found on mature diseased seedling limes, but not on affected plants in the nursery. Marked pitting is always present in diseased lime on rough lemon stock, young plants usually producing the typical vein clearing and vein flecking leaf symptoms, which simplify detection in the field. The indications are that infection increases in the open and with wider spacing as opposed to shade and closer spacing, a result presumably correlated with the behaviour of the vector. Affected Foster grapefruit on rough lemon shows pitting very distinctly, but Marsh on sour orange or rough lemon shows none, only occasionally a slight striation under the bark. Attempts at rehabiliation of the lime industry by use of rough lemon stock have not proved wholly successful, due to extensive suckering of the stock and comparative rapid reappearance of die-back contracted from neighbouring sources of infection. Cleopatra tangerine (mandarin) is to be tried as an alternative stock but it seems doubtful whether, with the common occurrence of the vector, any stock will protect the limes satisfactorily.

There has been a tendency to confuse the Gold Coast lime die-back with the die-back disease of lime in the West Indies. The two are demonstrably distinct. The West Indian disease starts from the root and collar and works up the stem, in the lower portions of which disease symptoms can always be found, below the die-back. In the Gold Coast disease the opposite occurs, and the lower portion of stems, the distal ends of which show marked die-back, are found to show no symptoms. In advanced stages of disease the ultimate appearance of dying trees may, of course, be superficially the same in both cases. Furthermore, if the virus were in fact present in the West Indies the occurrence of Aphis citricidus and of citrus on sour orange stock should result in widespread occurrence of the disease on other varieties of citrus.

It seems desirable to find a common name for the disease at present known as 'stem pitting', 'tristeza', 'quick decline' and 'Gold Coast lime disease', all now agreed to be essentially the same. Some such term as 'virus die-back of citrus' is suggested as possibly suitable.

Paper: Virus infections in South African citrus, associated with the disease of stem-pitting and tristeza.

by A. P. D. McCLEAN
(Division of Plant Pathology, Pretoria.)
[Presented by F. C. Loest, South Africa]

It is now accepted that virus infection is general in citrus grown in South Africa: a conclusion based on results of inoculations from orchard trees. The infection is graft-transmissible, readily perpetuated in daughter trees propagated vegetatively from diseased parents and is transmissible from diseased sources to healthy trees by at least one insect vector, Aphis citricidus. There is no evidence that it passes from diseased parents to their seedling progeny, whether the seedlings are of sexual or asexual origin.

NATURE OF THE INFECTION IN ORCHARD TREES

The following information on the nature of the infection is now available from the results of extensive transmissions from orchard trees to healthy citrus seedlings. Transfer of the virus was either by grafting or by means of the insect vector.

(1) The infection in orchard trees is not uniform and the virus content in different trees and particularly in trees of different species may differ qualitatively. To illustrate this a table is appended summarising the main reactions of a range of indicator seedlings to infections recovered by grafting from orchard trees of our main commercial species - sweet oranges, tangerine, grapefruit, and lemons. Attention is especially drawn to the difference in behaviour of some indicators to the infection from sweet oranges and tangerines on the one hand and to that from grapefruits and lemons on the other. The results follow closely those reported from Australia by Fraser[1], who ascribed the difference to an extra component in the "sweet-orange" or "tangerine" complex. She called it "seedling-yellows" virus. On the other hand the infections from the different sources resemble each other in producing symptoms of wood pitting in grapefruit and the West Indian lime, though differences occur in the severity of the diseases produced in these hosts.

(2) In most orchard trees the infection is not a simple virus but appears to be a complex of some kind. Evidence for this is the fact that sometimes aphids do not transfer the whole complex from a tree but only a part of it, which induces either a milder reaction or no reaction at all, depending on the species of the test seedling. For example, some isolates recovered from sweet oranges by aphids have lacked the "seedling-yellows" component and failed to induce a "yellows" reaction when transferred to sevilles. Other isolates have been of the simplest kind and capable of inducing only a very mild disease in the West Indian lime with no apparent harm to the seedling as a whole. A similar mild infection was found twice to occur alone in orchard trees: one in a Cecily grapefruit and the other in a variety of shaddock.

(3) When an infected graft is implanted in a healthy citrus seedling, the infection passes from the graft into the seedling itself whence it can be recovered by subinoculation. This holds true for most types of citrus with the possible exception of Poncirus trifoliata. But from observations on seedlings in the glasshouse, species vary in their behaviour to the infection from a given source. In sensitive species like the West Indian lime the infections from most sources produce a well defined disease with distinctive symptoms in the leaves and wood and accompanied mostly by a suppression of growth. At the other extreme highly tolerant species (sweet orange, tangerine and rough lemon) continue to grow at a normal rate without visible signs of harm. Symptoms are either absent or develop mildly. The behaviour of grapefruit is intermediate. When infected from grapefruit or lemon sources grapefruit seedlings continue to grow reasonably well with little outward signs of disease though the leaves may shew some clearing of the veins and the wood well defined pitting. When seedling-yellows is present in the complex the effects are more severe particularly in the early stages when growth is suppressed. Seville and lemon seedlings

[1] Fraser, L. Seedling yellows, an unreported virus disease of citrus. —Agric. Gaz. of New South Wales, 63, pp. 125-131, 1952.

TABLE: The behaviour of indicator seedlings to infections recovered from orchard trees.

SOURCES OF INFECTION (Orchard trees)	WEST INDIAN LIME	GRAPE FRUIT	SEVILLE & EUREKA LEMON	SWEET ORANGE (on own root)	SWEET ORANGE (on seville root)
Sweet oranges and tangerines	Severe disease: vein clearing, severe pitting, severe stunting, yellowing and distortion of leaves, extensive corky eruption on leaves.	Comparatively severe check on growth at first with formation of small, yellow leaves, followed by improvement in growth, cleared zones in veins, and extensive pitting	Seedling-yellows reaction: severe stunting, small yellow leaves, multiple bud formation, no pitting.	No check on growth, either no symptoms or a few cleared zones in the veins.	Severe tristeza reaction: rapid decline of scion, yellowing of leaves, and dieback.
Grapefruits	Mostly a disease of intermediate severity with clearing of veins, extensive pitting and occasional corky eruptions on leaves; sometimes a severe disease and sometimes a very mild one.	Usually some check on growth at first, followed by renewal of growth, few cleared zones in veins, extensive wood pitting.	No "yellows" reaction. No interruption of growth, and either no symptoms or cleared zones in veins of young leaves and sometimes wood pitting as well.	No check on growth, either no symptoms or a few cleared zones in the veins.	Comparatively slow decline of the scion; but in some cases the scion has continued to grow and its leaves have remained green.
Eureka lemons	Mostly a disease of intermediate severity with clearing of veins, extensive pitting, and occasional corky eruptions on leaves; sometimes a comparatively mild disease .	Usually some check on growth at first, followed by renewal of growth, few cleared zones in veins, and wood pitting.	No "yellows" reaction. No interruption of growth, and either no symptoms or cleared zones in veins of young leaves and sometimes wood pitting as well.		No visible reaction, growth of scion not interrupted, and leaves remain green.
Lisbon lemons	Comparatively mild disease with clearing of veins and wood pitting.		No "yellows" reaction. No interruption of growth and no symptoms.		No visible reaction, growth of scion not interrupted and leaves remain green.

are apparently less sensitive than grapefruit to infections from grapefruit and lemon sources but even more sensitive than grapefruit to ones containing the "yellows" component.

The behaviour of Poncirus trifoliata to infection is not fully understood. We have been unable to recover virus from seedlings into which infected grafts have been established; nor have we recovered anything from shoots that have developed from Trifoliata rootstocks of orchard trees although virus was present in the scions. But well-defined pitting has been observed in the wood of Trifolata stocks under the scions of both grapefruit and sweet orange.

When an infected graft was inserted into the end of a trifoliata branch growing on a lime seedling the infection passed through the trifoliata into the lime although it was not recoverable from the intersect.

(4) When one species of citrus is grafted on the roots of another to form a composite tree (which is the practice in commercial propagation), the behaviour of the tree as a whole to virus infection is influenced not only by the sensitivity of the scion but also by the sensitivity of the rootstock. Sweet orange seedlings on their own roots exhibit marked tolerance to the infections from all sources. If scions from such seedlings are grafted on the roots of another tolerant species, like the rough lemon, the tolerance of the tree as a whole remains unaltered. But if the tolerant scions are put on Seville rootstocks and infection is introduced by grafting from a sweet orange or similar source a marked change occurs in the tree: the scion ceases to grow or growth is much retarded, its leaves turn yellow and the branches die at the tips. These secondary effects, which reflect in the scions, are the result of direct injury to the species forming the rootstock. It is of special significance to us that a complex containing "seedling-yellows", to which Sevilles are particularly sensitive, is the one that produces the most severe and most rapid decline of the scion in composite trees with Seville as the rootstock. This probably also holds true for trees with Eureka lemon as a rootstock. The effects of isolates from grapefruit sources on composite trees of the same combination have varied: some caused a decline of the scion but one less severe and slower in its action, and others have had little harmful effect on the scion, which has continued to grow. Isolates from lemon sources have also had no apparent harmful effects on sweet orange scions over seville stocks.

(5) A new virus (vein enation virus) was found to occur with the stem-pitting complex in some orchard trees, including sweet oranges, grapefruits, and lemons. It is transmissible by grafting and by Aphis citricidus. When isolated in pure culture and transferred to citrus seedlings the virus produces small swellings or enations of the veins on the undersurface of some leaves but without doing any perceptible harm to the seedlings. When it is transmitted together with a stem-pitting complex the vein swellings are usually inhibited if the species of test plant is sensitive to the stem-pitting component.

EFFECTS ON CITRUS IN SOUTH AFRICA

So far as is known, the infection has had no adverse influence on the growing of sweet oranges. This section of the industry has continued to be productive and commercially sound. The reason must be attributed to the use of rough lemons as a rootstock and to the tolerance possessed by the

rough lemon and the two principal commercial varieties of sweet orange, Valencia and Washington Navel. Although the vigour and growth of young orange trees is apparently unaffected by infection it is possible that ultimately the tree is harmed and its productive life shortened. There is evidence for this in a tendency for a premature decline of orange trees in older orchards of 25 years or more. Neither rough lemons nor sweet oranges are completely devoid of symptoms, and a mild form of wood pitting is common in many trees; while in some varieties of oranges being grown experimentally wood pitting is extensive.

In grapefruits the infection produces the disease known as stem-pitting, which is ultimately accompanied by a severe decline of the tree. A similar disease with symptoms of wood pitting and secondary decline has been observed in experimental plantings of the citron, Tahiti lime, and the West Indian lime.

Budded trees of the Natal tight skin naartjie (variety of tangerine) on rough lemon stock have a commercial life of no more than 10 - 12 years. But seedling trees of the same variety continue to be productive after 30 - 40 years. The exact cause of the early decline and severe die-back of budded trees has not been established, but it is possibly associated with the infection known to be present in the trees.

Eureka lemons on rough lemon stock also tend to decline after a certain age. This may be a further example of the harmful effects that eventually overtake the ageing tree in spite of good tolerance of the tree when young.

In field experiments some species of citrus have been unsatisfactory as rootstocks, and scions of commercial varieties have either failed to grow on them or the resulting trees have been poor and unthrifty. That some of these failures are primarily caused by virus infection is now established. The best example is the Seville orange. Its apparent incompatability with some species of citrus - especially the sweet orange, tangerine, and grapefruit - has been known in South Africa a long time. But only of recent years has the incompatability been shown to be caused by the sensitivity of the Seville itself to virus infections carried in the scions. The degree of incompatability varies with the different species used as scions. The highest degree of incompatability is shown in the the combinations with sweet orange and tangerine. Grapefruit scions make a slightly better performance but not sufficiently to develop a productive tree. But some varieties of lemons (Eureka and Lisbon) grow well on Sevilles and develop into sizeable trees in spite of the infection carried by them. The simple explanation for the variation is that the infections carried in the scion differ qualitatively, and that the Seville is most sensitive to the infection in sweet oranges and least sensitive to the one in lemons.

Three more examples of rootstocks, which have been failures are the sweet lime, sweet rough lemon, and Poncirus trifoliata.

RELATION TO INFECTIONS IN OTHER COUNTRIES

Virus infections similar to those in South Africa are reported from other parts of Africa, Australia, the East Indies, and the Americas. Two important diseases are attributed to them; tristeza (or quick decline), a disease of citrus trees (mainly sweet oranges) on Seville stocks in South America and California, and lime die-back, a disease of seedling trees of the West Indian lime in the Gold Coast Colony. A disease of grapefruits similar to

stem-pitting is reported from Australia and the Argentine. That there is a relationship between the causal viruses of these diseases is suggested by a similarity in the behaviour of indicator species to the infections and by the ability of the infections to produce symptoms of wood-pitting in species like grapefruit and the West Indian lime. But there are probably differences just as there are qualitative differences in the infections occurring naturally in the citrus clones grown in Australia and South Africa. If the cause of tristeza is the same as the complex carried latently in South African sweet oranges and tangerines, and the experimental evidence supports this, then it is wrong to say that tristeza and stem-pitting are synonymous and caused by the same virus. The complex occurring in diseased grapefruit in South Africa and Australia lacks the "seedlings-yellows" component present in the sweet orange complex, and for this reason tristeza and stem-pitting are not homologous.

Xyloporosis, a disease of citrus trees on sweet lime stock in Palestine, has a resemblance to the disease associated with the stem-pitting - tristeza complexes. The symptom of wood pitting follows much the same pattern. The suggested relationship has been opposed by some authorities on the score that the Seville is insensitive to xyloporosis and that the condition of diseased trees is improved by inarching to Sevilles. But this may mean that the infection in Palestine lacks the seedling-yellows component. From a recent report by Reichert[2] it would appear that even the Seville is not completely insensitive. He states that Seville rootstocks sometimes shew a type of wood pitting and that the scions often shew little leaf symptoms and a slow decline as well. But the nature of xylorporosis and its relationship to stem-pitting and other infections will remain unexplained until proper transmission experiments have been undertaken in Palestine.

CONTROL

Practical means of eradicating this group of infections once it has gained a foothold is unlikely to succeed in any country possessing an insect vector. Moreover, such factors as spread of infection by aphides, the perpetuation of infection in the clonal progeny of infected parents, and the fact that the host is a tree with a semi-permanent place in the field mean that the infection will ultimately become general in citrus trees. This is the position for South Africa.

The growing of sweet oranges presents no special problem. The sweet orange is itself a highly tolerant species and can be grown successfully if trees are raised on a tolerant rootstock. But a tolerant stock is not the complete answer to the problem as a whole because the decline of some citrus trees is caused by direct injury to the scion itself. This is the case in the more sensitive grapefruit, which declines at a comparatively early age even when budded on a tolerant stock. The main problem for the future is how long existing citrus clones, including those of sweet oranges, will continue to be productive under the influence of virus infection. The solution probably lies in the nucellar seedling.

Most citrus species produce through their seed a high proportion of nucellar seedlings, which, because of their asexual origin in the nucellus, conform closely to the parent type. They possess juvenile vigour and initially are free of infections that may be present in the parent. Work in

[2]Reichert, I. Xyloporosis in citrus. Report of thirteenth Internat. Hort. Conf. 1952.

California by Frost and others indicates that the nucellar seedling provides a means of re-establishing commercial varieties when they become weakened by virus disease or old age. Objections to its immediate use as an orchard tree are raised on the grounds of thorniness, slowness in coming into bearing, and the uncertainty of its ultimate performance from a commercial standpoint. But experience has shewn in California and elsewhere that nucellar seedlings produce vigorous trees which in many cases bear good crops of quality fruit.

In South Africa the problem of early decline in the Natal tight skin naartjie when budded on rough lemons has been solved by growing this variety as seedling trees on their own roots. In recent years seedlings have been planted extensively in some districts. Their performance is much superior to that of budded trees. The fruit is of good quality and shews a high degree of uniformity. Preliminary observations from a small experimental planting of grapefruit seedlings suggests that the same procedure may be successful for grapefruit as well.

Some species, like the West Indian lime, are too sensitive to be grown directly on their own roots, but there is evidence that by top working seedlings to rough lemon stocks the tree acquires better tolerance. This procedure is being followed in the Gold Coast in an attempt to resuscitate its lime industry.

Mild strains of virus are reported from most countries. They offer a possible means of protecting sensitive trees against the harmful effects of the full virus complex. But it is still a matter for experiment whether this method of immunization has practical possibilities.

Paper: Tristeza stem pitting in New South Wales

by LILIAN FRASER
[Presented by C. J. Magee, Australia]

Evidence of early presence: In the late 1860's citrus in New South Wales, which was well established around Sydney, was devastated by an outbreak of collar rot (now known to be due to Phytophthora citrophthora). The use of Seville stock had saved the citrus industry in Spain when a similar disease outbreak occurred about 20 years previously and the introduction of Seville stock was pressed by the Government Botanist who had visited Spain to examine the disease. However, the introduction of Seville stock to New South Wales was not successful. It was stated in contemporary literature that trees on this stock would not grow and records of nurserymen who tried to work the stock with currently-grown varieties of orange show that they were unsuccessful. This is taken as evidence that the tristeza virus was well established in New South Wales citrus by about 1870. Aphids, the vectors of this disease, were also present by that time.

Present surveys, Coast: In New South Wales citrus is grown in several coastal districts under moderate rainfall conditions and aphids are numerous at most times of the year. Surveys over the past three years, using seedlings of West Indian lime and Seville orange as indicators, have shown that tristeza is universal in all citrus varieties. A number of strains have been obtained differing in severity of symptoms produced on West Indian lime.

Inland: Citrus is grown in a number of irrigation districts in western New South Wales under arid conditions and high summer temperatures where aphids are of relatively rare occurrence. Some orange and grapefruit varieties were introduced into the Mildura area direct from California about 60 years ago and propagated locally or in South Australia. A proportion of their progeny is still free of tristeza. Many trees, however, were also brought in from New South Wales coastal nurseries and in recent surveys about 70 per cent. of the orange and grapefruit were found to be infected. Slow spread of stem-pitting is occurring through grapefruit orchards.

Introduction to New South Wales: Tristeza could have been introduced in budwood or living trees from South Sfrica in the early days of settlement. It is unlikely that the virus is endemic in New South Wales. A few specimens of native citrus from north coast rain forest areas (Microcitrus australasica) and north-western plains (Eremocitrus glauca) so far tested have been found to be free of tristeza, but susceptible to it. No native aphids occur in Eastern Australia.

Spread from New South Wales: Trees from Sydney metropolitan nurseries have been distributed to all parts of Australia. In 1931 a shipment of 300 trees was sent to a grower in the Argentine. Tristeza is believed to have started in Argentina about 1933, and thereafter spread with increasing speed throughout Argentina to Brazil.

Effect on orange and mandarin industry in New South Wales: Because of its early failure no trees are grown on Seville stock in New South Wales. Field observations and glasshouse trials to date indicate that the virus has no adverse effect on trees on rough lemon or sweet orange stocks, which are tolerant, or on Poncirus trifoliata stock, which is immune from tristeza.

Effect on grapefruit industry: No extension of grapefruit planting is going on in New South Wales at present. Many trees are known which show no symptoms of disease but carry the tristeza virus. The possible protective value of mild virus strains is under trial.

Discussion: DR. SILBERSCHMIDT said he was interested in the history and discovery of the tristeza disease. He was under the impression that it came from South Africa to the Argentine and then Brazil, where it killed practically all the orange trees growing. It was now present in South Africa, Australia, and New Zealand, and he was astonished that any oranges still existed in these countries.

MR. BAWDEN asked MR. W. A. HUGHES whether he had ever transmitted the same virus from lime seedling to lime seedling by both mealy bug and aphids.

MR. W. A. HUGHES replied that using experimentally graft-infected (not naturally infected) lime seedlings both die-back and little leaf viruses had been transmitted to healthy plants by the mealy bugs and aphids. He added that similar results had been obtained using naturally infected trees in the field as source plants.

MR. BAWDEN agreed that something had been transmitted, but was not satisfied of positive evidence that both insects has transmitted the same virus.

MR. W. A. HUGHES said that similar source plants were used for both insects and similar symptoms were produced in different test plants.

MR. BAWDEN did not accept this as proof, and asked whether each virus had been transmitted by one kind of insect to a healthy seedling and then carried on by the other kind of insect to the next healthy seedling. Other than that he could not accept that the two viruses had been transmitted by the same insect.

MR. W. A. HUGHES said by that criterion they had not.

MR. BAWDEN said that there was no other criterion.

MR. LISTER asked DR. WATERSTON if the shade effect on symptom expression in infected limes had been measured with any degree of accuracy.

DR. WATERSTON was of the opinion that the figures represented were observation.

MR. BAWDEN asked what was meant by shade.

MR. WATERSTON said that no special trees were planted as shade trees for the crop. They were forest trees.

MR. W. A. HUGHES said that by "shaded" he meant that the forest trees were not felled and smaller trees were planted in an endeavour to provide additional shade, but the actual amount of shade was extremely light compared with what had just been mentioned.

MR. BAWDEN suggested it might not be a shading effect at all.

MR. HUGHES disagreed saying that the presence of the forest and smaller trees made a considerable difference in the Gold Coast, where sunlight was intense.

MR. BAWDEN said the effect of big forest trees on the dispersal of insects must be enormougly great compared with reduction in light intensity.

by C. G. HUGHES
(Bureau of Sugar Experiment Stations, Brisbane, Queensland)
[Presented by C. J. Magee, Australia]

Ratoon stunting disease in the sugar world is a subject for research and discussion amongst pathologists, agriculturalists, plant breeders, and administrators for its role as a possible factor in the "running out" of varieties makes it of importance to all connected with the production of cane.

It has long been accepted that plants propagated vegetatively suffer deterioration or loss in cropping power and the sugar industry in all countries has had to use a continuous stream of new varieties to maintain production per acre as established varieties failed. At the present time the decline of the commercial varieties has in many cases proved to be due to ratoon stunting disease and it may be that much of the decline recorded in the past had the same cause. It is not suggested that all instances of decline are due to this disease but evidence has indicated that the trouble is responsible for the abandonment of many varieties in Queensland, where the disease

was first discovered.

The disease is described as virtually symptomless since it has no obvious external symptoms except a stunting and loss of vigour, which are particularly marked in the ratoon crops, and when growing conditions are harsh. Concomitantly with the stunting is a very slow early growth in the ratoons, an undue susceptibility to dry weather, and, should drought conditions prevail, actual death of the stools. The stunting is not as severe in plant crops as in ratoons and even in the latter may be considerably reduced by adequate watering throughout the crop. A large range of varieties is known to be infected in Queensland and many of the canes suffer severe losses such as a reduction in the ratoon crop from 25 tons for healthy to 9.7 per acre for diseased.

The internal symptom of ratoon stunting (which prevents the disease being referred to as symptomless) takes the form of a discoloration of vascular bundles at the base of the mature nodes. Even this symptom, however, cannot be claimed as being unique for ratoon stunting since several other diseases, e.g., leaf scald and chlorotic streak, produce almost identical effects, although in these instances external symptoms are present to confirm the diagnosis. The discoloration is reddish yellow and can best be seen when the stalk is sliced longitudinally with a sharp knife. A dull knife tends to split rather than cut and the colour in the vascular bundles is then not so easily seen. Bundles near the surface of the stalk should be disregarded when looking for ratoon stunting since infection from outside frequently discolours them. The number of bundles affected may vary greatly from one or two per node to such a number that the impression of a reddish zone is given, and for positive identification it is advisable to look at several nodes in the single stalk, since unexplained discolorations occur in healthy ones. The reddening is most obvious in the central portion of mature stalks but there appears to be no constant pattern as to the time of year when symptoms may first be seen. Young nodes are not suitable for examination since they darken rapidly on exposure to the atmosphere and the basal nodes are unreliable because of the chance of discoloration arising from other damage. Sections through discoloured bundles usually show a clogging of the xylem vessels with a red dark amorphous mass; sometimes phloem necrosis can also be seen.

Efforts to find the causal agent of ratoon stunting have been fruitless and the electron microscope has failed to reveal any virus particles. Preliminary experiments in the production of an antibody in rabbits have given indication of a reaction but it was not sufficiently definite to allow its use in diagnosis. The evidence at present available indicates that the disease is caused by a virus.

Ratoon stunting can be readily transmitted mechanically by inoculation of the sett or the growing stalk; attempted inoculations through leaves of the apical growing point were not successful. Leaf and stem juice is highly infectious and the latter may be diluted at least 10,000 times without any loss in infectivity. Setts may be inoculated by pressure into the cut ends, by dipping the freshly cut ends, or by cutting with a knife contaminated with infective juice. Infection of the growing stem was obtained by hypodermic needle. In the field the most important method of spread is the cane knife used in harvesting or the knife or cutter planter blade used in cutting setts for planting. The disease is not transmitted through the soil although there have been a few instances of unexplained natural spread of the disease from diseased to healthy plants in areas subject to waterlogging.

The very small amount of natural spread, which is in marked contrast to the rapid spread of chlorotic streak disease, is an invaluable aid in the control of ratoon stunting. It means that stocks of clean plants may be built up in commercial cane areas without the necessity for any special precautions except the sterilizing of knives and cutter-planters. In many districts stocks of some canes have not become saturated with the disease and what appear to be clean sources are still available. However, even these are not above suspicion since the previous history of the blocks is not always known, and it is fortunate indeed that disease-free plants can be obtained by heat treatment of the setts. Experiments with the very susceptible Q. 28 demonstrated that with this variety the disease could be controlled by immersion of the setts in water at 50°C. for 1½ hours or 52°C. for one hour; more severe treatments also gave control but the setts suffered damage and some poor germinations resulted. Subsequent extension of the work to include a number of varieties at different times of the year on a commercial scale showed that all varieties were not as resistant to the treatment as Q. 28, and 50°C. for two hours is now recommended for general field purposes. Even with this treatment some varieties are unreliable in germination but generally, providing succulent plants are avoided, good stands are obtained. The physical problems presented by the treatment of heavy and bulky quantities of clean plants are considerable but tanks and auxiliary equipment have been set up in all districts, and in some the goal of having all disease-free plants for the 1954 planting is already in sight. In one district, for instance, sufficient setts were treated to plant 250 acres, which under irrigation will yield 10,000 tons of plants for this season. The treatment of setts in air at 53° to 54°C. for eight hours also renders them disease-free but is not as easy to handle as hot water. In Louisiana, where only succulent material is available for plants, a large semi-trailer has been fitted as an experimental, mobile, hot air treatment outfit.

Ratoon stunting was first discovered in the Mackay district, 600 miles to the north of Brisbane and 2° within the tropics, early in 1945 after a dry spring had caused a disastrous drop in ratoon tonnages, but the lack of obvious symptoms and the lag in obtaining information due to having to wait for ratoon crops meant that it was not until 1952 that a complete survey revealed that the disease was present in all cane-growing districts of Queensland. Specimens brought from the neighbouring state of New South Wales were found to be infected and overseas visits by Bureau officers during 1953 revealed the disease in Hawaii, United States of America, British West Indies, Colombia, and South Africa. In addition, there is evidence that it is also present in India, Mexico, and Puerto Rico.

Paper: Some aspects of the heat treatment of sugar-cane planting material

by P. B. HUTCHINSON & W. L. WISE
(Imperial College of Tropical Agriculture)
[Presented by R. M. Nattrass, Kenya]

SUMMARY*

If maximum germination is required there are many factors which should be taken into account before postulating a heat treatment to control virus

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* This work was done jointly by the Department of Botany and Plant Pathology and the British West Indies Sugar Research Scheme.

diseases in a particular area. Of these, the variety concerned is possibly the chief factor and it is likely that some varieties are sufficiently sensitive to necessitate special attention to other factors if adequate germination is to follow severe heat treatments. In this case the advantages of precisely controlled treatments are obvious and hot water appears preferable to hot air in this respect. It is possible also that factors such as the part of the stalk used for planting material, its diameter, and the depth at which it is planted would have to be controlled.

Evidence is given that the presence of chlorotic streak disease lowers the germination of planting material and also renders it more intolerant to heat-treatment than non-infected material. There is also tentative evidence to support the view that ratoon stunting disease does not inhibit germination.

Paper: Ratoon stunting disease in Natal

by N. C. KING
(S. A. Sugar Association Experimental Station, Mt. Edgecombe)
[Presented by G. R. Bates, Southern Rhodesia]

Sugar-cane has been cultivated for a long time in Natal and there has been a constant change of varieties due to loss in yielding power, now thought to be caused in part at least by ratoon stunting disease. But before dealing with ratoon stunting disease, let us consider the varietal position in Natal.

The variety Uba, which was at one time exclusively grown, was extremely susceptible to a virus disease, streak, which reduced the crop by 12 per cent. It was replaced by some of the more highly complex hybrids, notably the P. O. J. and Co. varieties but the position to-day is that we find hardly any of the P. O. J. and Uba varieties grown, and Co. 281 in many areas is yielding so poorly that it is rapidly going out of cultivation. It is known why the P. O. J., Uba and some Co. varieties have been discarded but as yet no valid reason can be given for the deterioration of Co. 281, investigations into which began in 1948.

Consider now the position of Co. 281 in the economy of the sugar industry over the last twenty years. In 1934 this variety was distributed to the planters and by 1944, 69 per cent. of the cane milled was Co. 281. This was the peak period and was maintained more or less at the same level until 1947 when the percentage of Co. 281 milled started to decline. By 1953 only 5. 7 per cent. of the cane milled was Co. 281. By this time, however, another good variety, N:Co. 310, had been distributed and may be partly responsible for discarding Co.281 but it does not account for the ploughing out of thousands of acres.

Experimental work on this important subject of the failure of Co. 281 was started in 1948. In a preliminary investigation good seed cane was obtained from a field in an area where Co. 281 was still growing fairly vigorously. The poor seed cane was taken from a ratoon crop of Co. 281 which had deteriorated rather rapidly. The two types of seed cane were then planted in a properly replicated trial.

From an analysis of the yield figures it was shown that a significantly higher yield was obtained from the good seed in the plant and 2nd ratoon crops. In the 1st ratoon crop there was no significant difference between the two types of seed cane but the indication was in the same direction. Ratoon

stunting disease has been found to be very prevalent in this field and during harvesting operations no attempt was made to sterilise the cane knives. It seems apparent, therefore, that by this time the whole experiment is infected with the disease.

In December 1952 seed cane was obtained from the aforementioned experiment. This seed cane was obtained from stools which had developed badly in the poor plots and from good stools in the good plots. Some of this seed cane was cross inoculated using the poor seed, showing symptoms of ratoon stunting disease, as a source of inoculum. Other seed cane was treated against chlorotic streak at 52°C. for 20 minutes and then planted. This experiment has not yet been harvested but the hot-water treated plots appear to be slightly better than the other plots.

The research workers at the Queensland Bureau of Sugar Experiment Stations were the first to associate a disease with the stunting observed in the variety Q. 28. As a result of the work carried out in Australia it was established that the presence of reddened vascular bundles in the region of the nodes indicated the presence of the disease. When this information was available a survey of the cane-growing area was carried out. This survey established the fact that the disease is widespread and no areas appear to be free from it. In carrying out this survey it was noticed that all varieties did not show the same type of symptom. In some there were a row of brown dots at the node and in others there were definite reddish-orange streaks. All the varieties commercially grown exhibited symptoms but in the variety N:Co. 310 they were more difficult to find. Whether this was due to the varietal resistance or to late infection is not certain. During this survey it was found that the symptoms were more easily located in poorly grown cane than in well grown cane and on the proper alluvial flats it was very often difficult to find the symptoms.

During the examination of varieties it was found that a number of seedlings supposed to be virus-free showed symptoms of ratoon stunting in two consecutive nodes. The presence of the disease in seedlings shows that a vector may be present since the seedlings have not yet been cut. This is now being investigated by the Entomologist.

The nodal discoloration is due to the formation of a substance (probably gum) which blocks both the xylem and phloem. This blocking usually takes place at about the position of the wax ring where the vascular bundle branches to the leaf sheath. The branches are apparently affected first and later the whole junction becomes blocked, including the main bundles of the stem itself. This would naturally cause the plants to show drought symptoms, such as the rolling and upright growth of the leaves, which can only be viewed with suspicion.

Since ratoon stunting disease has been found in Natal the question naturally arises as to what loss in yield is taking place and what future loss in crop can be expected. It must be realised that the presence of the disease has only definitely been established in Natal for little over a year and naturally the experiments planted to test the varietal reaction have not had sufficient time to mature and be harvested. However, we have noticed fields of N:Co. 310 and Co. 331 which are extremely uneven and the symptoms of the disease are more easily found in the stunted cane than in the better grown stools. If the unevenness is due to the disease only, then one can anticipate a fair loss under adverse growing conditions. The distribution of the variety

N:Co. 334 has been held up due to the very uneven stand of cane and profuse production of ratoon stunting disease symptoms when growing on a poor shaley soil. It is felt that it is better to withhold it than to allow the planters to cultivate a variety which may show considerable loss due to ratoon stunting when growing under adverse conditions.

The work of the Queensland Bureau of Sugar Experiment Stations has shown that the disease can be cured by treating the setts prior to planting with hot water. At Mount Edgecombe it was found, as was to be expected, that all the varieties did not react in the same way. Some gave good germination after being treated for 54°C. for $1\frac{1}{4}$ hours, while others failed to germinate. It must be realised that the temperature required to suppress ratoon stunting disease is very near the thermal death point of the cane and in some this is exceeded when giving the hot water treatment at 54°C. Fortunately a lower temperature for a longer time also cures the disease, and 50°C. for 2 hours has been recommended to our planters; although not as much cane can be treated in a given time it nevertheless assures a reasonable germination. After heat treatment the setts are more prone to attack by micro-organisms and should consequently be completely dipped in a fungicidal solution. The conditions for germination should be as near the optimum as possible so as to allow the shoots to emerge without delay.

During the hot-water treatment of sugar-cane setts it was found that the pure Saccharum officinarum varieties germinated very poorly. The age of of the buds seemed to make no difference and in order to establish these varieties the hot-air treatment of 50°C. for 24 hours was given. Germination was better but still far from satisfactory. The Sugar Experiment Station at Mount Edgecombe is recommending that the cane growers should treat enough cane by heat to establish small plots of the varieties which are most suitable for their particular farm conditions. These plots can be extended by using seed cane obtained from the original nursery. It is not known what loss ratoon stunting is causing but it is felt that if there is a big loss then measures are already under way to control the disease.

DR. BATES concluded by saying that this particular disease, by showing no obvious symptoms, made clear the limitations of quarantine.

MR. BAWDEN asked why hot-water treatment, which was used for treating sugar-cane setts as long as 30 or more years ago, had suddenly regained popularity. Was it because of something other than ratoon stunting?

DR. MAGEE said that heat treatment had been used in Queensland for many years against chlorotic streak and had become the standard practice among the producers of planting stock. He thought that it might also be in use in Louisiana and Hawaii, as it was a suitable method for the type of grower in those areas.

MR. BAWDEN replying to a question as to whether virus inactivation is claimed said that treated cane produced healthy plants, but the interpretation of that was optional. There had been a suggestion that hot-water treatment was, in fact, selection by killing infected stock.

DR. PRENTICE said he wished to emphasize the difficulties of employing quarantine as a means of preventing the spread of latent viruses because spread had often occurred before they had been recognised. There were no sure methods of detecting them as many of the viruses and suitable indicator plants were unknown. He did not think that with our present knowledge the solution

of the problem lay in quarantine and testing.

DR. MAGEE agreed that diseases had been spread round the world in this way and was aware that many more would follow unless quarantine could be better organized. He greatly feared the introduction into Australia of Pierce's disease of grape vines, which precluded the cultivation of the vinifera grape in Florida. Grapes were an extremely important fruit crop in Australia. The virus of Pierce's disease had a very wide host range and could be introduced to other important grape-growing countries, such as South Africa and France, on any one of these hosts, although it would be officially excluded in vine plants. This was only one instance and he felt that the Conference could do little more than draw the attention of the quarantine authorities to the seriousness of the position.

MR. BAWDEN agreed that more should be done, but every alternate host could not be tested otherwise plant movement would cease altogether. He intimated that more care should be utilised by plant breeders in collecting their material from other countries.

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